

VOLUME 9

NUMBER 1

ARCHIVES OF NEUROLOGY AND PSYCHIATRY

EDITORIAL BOARD

T. H. WEISENBURG, Philadelphia

ALBERT M. BARRETT, Ann Arbor, Mich.

HUGH T. PATRICK, Chicago

SAMUEL T. ORTON, Iowa City

E. W. TAYLOR, Boston

FREDERICK TILNEY, New York

JANUARY, 1923

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH
DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, \$6.00

Entered as second-class matter, Jan. 7, 1919, at the postoffice at Chicago, Illinois, under the Act of
March 3, 1879. Acceptance for mailing at special rate of postage provided for
in Section 1103, Act of Oct. 3, 1917, authorized Jan. 15, 1919.

CONTENTS OF PREVIOUS NUMBERS

OCTOBER, 1922. NUMBER 4

Effects of Antisyphilitic Therapy as Indicated by the Histologic Study of the Cerebral Cortex in Cases of General Paresis. A Comparative Study of Forty-Two Cases. H. C. Solomon, M.D., Boston, and A. E. Taft, M.D., Philadelphia.

Progressive Funicular Myelopathy (Subacute Combined Degeneration). Joseph H. Globus, M.D., and Israel Strauss, M.D., New York.

Papilloma of the Fourth Ventricle: Report of a Case. Ernest Sachs, M.D., St. Louis.

Magnus and De Kleijn Phenomena in Brain Lesions of Man. A Consideration of These and Other Forced Attitudes in the So-Called Decerebrate Man. I. Leon Meyers, M.D., Los Angeles.

Diagnostic Value of Blood Sugar Curves in Neurology. Sidney I. Schwab, M.D., St. Louis.

Malignant Tumors of the Nasopharynx with Involvement of the Nervous System. Henry W. Wolman, M.D., Rochester, Minn.

Abstracts from Current Literature:

Dementia Asiatica (Bianchi's Disease).—Seriation.—Physiology and Pathology of Tickling.—Pathological Studies in Dementia Praecox.—Pathology and Treatment of Spinal Cord Tumors.—Field Defects Produced by Temporal Lobe Lesions.—Cerebral and Unilateral Fever.—Sequelae of Epidemic (Lethargic) Encephalitis.—Pathogenesis of Disturbances of Sleep Following Encephalitis Lethargica.—Results of Fifty Decompressions for Epilepsy.—Mongolian Idiocy in a Chinese Boy.

Society Transactions:
Philadelphia Neurological Society.
Book Review.

NOVEMBER, 1922. NUMBER 5

An Analysis of Fourteen Consecutive Cases of Spinal Cord Tumor. Charles H. Frazier, M.D., and William G. Spiller, M.D., Philadelphia.

The Mechanical Effects of Tumors of the Spinal Cord: Their Influence on Symptomatology and Diagnosis. Charles A. Elsberg, M.D., and Byron Stookey, M.D., New York.

Shall We Decompress for Choked Disk? B. Sachs, M.D., New York.

Results of the Removal of Tumors of the Spinal Cord. Alfred W. Adson, M.D., and William O. Ott, M.D., Rochester, Minn.

Dystonia Musculorum Deformans with Especial Reference to a Myostatic Form and the

Occurrence of Decerebrate Rigidity Phenomena: A Study of Six Cases. I. S. Wechsler, M.D., and S. Brock, M.D., New York.

Abstracts from Current Literature:

Decerebrate Rigidity in Man and the Occurrence of Tonic Fits.—Anatomic Conditions of Binocular Vision.—Pathology of Psychoses of Senility.—Transorbital Puncture of Gasserian Ganglion.—Psychologic Inquiry into the Nature of the Condition Known as Congenital Word Blindness.

Society Transactions:
American Neurological Association.
Chicago Neurological Society.
Book Reviews.

DECEMBER, 1922. NUMBER 6

Recent Studies on Spirochetes in General Paralysis. Charles B. Dunlap, M.D., Ward's Island, N. Y.

An Anatomic Study of the Faisseau de Tüsch in Relation to the Temporal Lobe. John H. W. Rhein, M.D., Philadelphia.

Tuberous Sclerosis. Walter Freeman, M.D., Philadelphia.

Disturbances of the Respiratory Rhythm in Children. A Sequela to Epidemic Encephalitis. Harry L. Parker, M.B., Rochester, Minn.

Psychopathology and Organic Disease. Smith Ely Jelliffe, M.D., New York.

Postencephalic Deformities of Motion: A Lecture Illustrated by Motion Pictures. S. Philip Goodhart, M.D., New York.

Pyramidal and Extrapyramidal System Involvement in Epidemic Encephalitis. S. Brock, M.D., and I. Margaretten, M.D., New York.

The Stricerebellar Tremor: A Study of the Nature and Localization of the Combined Form of Organic Tremor. J. Ramsay Hunt, M.D., New York.

Abstracts from Current Literature:

Myelinization in the Cerebral Cortex.—Facial Paralysis.—Mental Hygiene.—Treatment of Severe Paraplegia in Pott's Disease by Puncture of Abscess Through Intervertebral Foramen.—Anhedonia.—Differential Diagnosis of Schizophrenia.—Diffuse Glia Reactions.—Auditory Zone in Man.—Comparative Studies in the Chemistry of Blood and Cerebrospinal Fluid.—Bilateral Cerebellar Abscess with No Localizing Symptoms.—Recklinghausen's Disease.—Bismuth in Syphilis.—Multiple Sclerosis in Children.—Diagnosis of Tumors

of the Cauda Equina, Conus and Epiconus Medullaris.—Concerning the Inferior Olive.—Treatment of Meningococcus Meningitis.—An Endocrine Factor in General Paresis.—Practice of Psycho-Analysis in a Public Clinic.—Necrinoma in Recklinghausen's Disease.—Meningitis Ossificans.—Compression Myelitis, Operation, Recovery.—Latent Time of Reflexes.—Etiology and Pathology of Chorea Minor.—Contagiousness of Epidemic Encephalitis.—Anatomic Studies of the Viscera in Dementia Praecox.—Latent Neurosyphilis.—Tumor of the Splenium of the Corpus Callosum.—Treatment of Antenatal and Congenital Syphilis.—Pseudotumor Syndrome.—Fracture Dislocation of the Spine Treated by Fusion.—Hypothyroidism.—Influence of Head and Body Posture on Spinal Fluid Pressure.—Hemorrhagic Meningo-Encephalitis in Anthrax.—Incidence of Hereditary Syphilis.—Methods for Bridging Nerve Defects.—Infantile Tetany.—Neurologic Significance of "Nonne Reactions."—Treatment of Syphilis by Mercury Inhalations.—Early Diagnosis in General Paresis and Tabes.—Tuberculous Meningitis.—Value of Ventriculograms in Localization of Intracranial Lesions.—Hypophyseal Obesity in Lethargic Encephalitis.—Intrathecal Tumors of Spinal Cord.—Functional Disease in Soldiers.—Kyphoscoliosis in Syringomyelia.—Late Hereditary Syphilis.—Spinal Caries and Compression Myelitis Lateral Sclerosis.—Congenital Goiter.—Syringomyelia.

Book Reviews.
Index.

Archives of Neurology and Psychiatry

Vol. 9

JANUARY, 1923

No. 1

SECTION OF THE ANTEROLATERAL COLUMNS OF THE SPINAL CORD (CHORDOTOMY)

DISCUSSION OF THE PHYSIOLOGIC EFFECTS AND CLINICAL RESULTS IN A SERIES OF EIGHT CASES *

CHARLES H. FRAZIER, M.D., AND WILLIAM G. SPILLER, M.D.
PHILADELPHIA

There are few conditions the physician has to treat which cause so much distress as that of intense pain uncontrolled by drugs. A means of relief in selected cases is afforded by chordotomy. The operation will not be entirely successful in every case in which it is employed—some pain fibers occasionally will escape the knife—but the operation has now been performed sufficiently often to justify a belief in its efficacy. It is a delicate procedure, requires a skilful technic, and is not to be undertaken lightly by one untrained in the surgery of the spinal cord. It requires on the part of the operator a visualization of the microscopic anatomy of the cord, and a realization that a slight misplacement of the incision may cause motor paralysis of one or both lower limbs, depending on whether the division is unilateral or bilateral. We¹ have written on this subject previously and therefore do not refer to the literature at the present time.

We report in this paper eight cases in which chordotomy has been performed. All but one have not been reported previously, but in this one case chordotomy for certain reasons mentioned in the notes has been performed three separate times on the same side of the cord. In one case (Case 8), the operation was performed to remove the intense hyperesthesia above the anesthetic area produced by transverse myelitis, and was entirely successful.

The operation abolished the pain for which it was performed in six of these cases, and gave decided relief from pain in the remaining

*Read at the Forty-Eighth Annual Meeting of the American Neurological Association, Washington, D. C., May, 1922.

1. Spiller, William G., and Martin, Edward: The Treatment of Persistent Pain of Organic Origin in the Lower Part of the Body by Division of the Anterolateral Column of the Spinal Cord, *J. A. M. A.* **58**:1489 (May 18) 1912. Frazier, Charles H.: *Surgery of the Spine and Spinal Cord*, New York, D. Appleton & Co., 1918, chapter 6. *Ibid.*: Section of the Anterolateral Columns of the Spinal Cord for the Relief of Pain: Report of Six Cases, *Arch. Neurol. & Psychiat.* **4**:137 (Aug.) 1920.

two cases. Four of these were cases of malignant tumor. In one of these, in which the suffering had been excruciating and constant, the relief was so intense that the patient expressed herself by saying she felt as though she were in heaven. In one case the childishness of the patient interfered with careful study, but there was reason to believe that much relief from suffering had been obtained. One case occurred in an army officer who had been injured in the war. Chordotomy was performed three times over a period of nearly three years. After the first operation relief from pain was great for a period of about three months. Whether the relief from this first operation was felt for a longer time than after the later incisions of the cord because the operation then was new to the patient, cannot be asserted, but this is possible. More than two years later the chordotomy was repeated with relief again, but not with complete cessation of pain. It was then found that certain fibers innervating a small strip on the inner side of the leg below the knee and on the inner sole of the foot, that is, in the distribution of the long saphenous nerve, must have escaped in the section of the cord, as it was possible to produce pain here by pressure, but the patient could not localize the pain so produced to the part irritated.

The long saphenous nerve was cut to determine whether the pain was of peripheral origin and could be relieved without further operation on the spinal cord. For a short time this procedure relieved the pain. A third chordotomy was performed lower in the cord, at the tenth thoracic segment, in the hope that if any fibers had escaped in the two previous chordotomies, they might occupy a different situation lower in the cord and be caught by the section of the anterolateral column here. The persistence of pain in a modified form in this case was so unlike the results in the other cases that we were perplexed. Certain observations convinced us that a considerable psychic element was present. It was found that sugar given at certain times to relieve pain did so promptly when the patient believed he was obtaining more powerful remedies.

We had no reason to believe that hemorrhage occurred into the spinal cord after chordotomy in any case, except possibly in one, in which some transitory paresthesia occurred in the foot three or four days after a second chordotomy and some transitory dysesthesia and blunting of temperature sensation but not of pain sensation in the lower limb after the third chordotomy, on the side of the operation. Whether this sensory disturbance on the side of the operation was caused by hemorrhage or edema or by some other lesion, we have no means of determining, but is probable that slight transitory impairment of function was caused in the side of the cord opposite to the chordotomy at the time of the operation by the necessary manipulation.

We feel convinced that no pain or temperature fibers, however, ascend in the spinal cord uncrossed, as in two other cases in which unilateral chordotomy was performed no disturbances for these forms of sensation developed in the lower limb on the side of the operation. On this side, pain, heat and cold sensations were normal after unilateral chordotomy. We feel convinced that this operation, either unilateral or bilateral, does not distinctly impair tactile sensation in the lower limbs, and therefore our observations confirm the opinion that for impairment of tactile sensation the lesion must destroy more than the anterolateral columns.

We are convinced that this operation when successfully performed destroys pain, heat and cold sensations in the lower limbs and lower part of the trunk when performed at the proper level, although the impairment of these sensations is less intense over the abdomen in some cases, probably because some fibers have not decussated at the level of the incision. In some cases we have observed that certain fibers innervating the lower limbs for pain and temperature sensations escape in the division of the anterolateral columns. This is not surprising, because in the endeavor to spare the crossed pyramidal tract it is possible that a few sensory fibers for pain and temperature escape.

We are impressed by the fact that the bladder and rectum almost invariably escape damage in this operation. In only one of these cases (Case 3) in which a more liberal section of the cord was made deliberately, was there loss of bladder control while the patient was in the hospital; in this case, too, partial paralysis of the lower limbs occurred, probably as a result of injury of the crossed pyramidal tracts. One might possibly expect sympathetic fibers for the bladder and rectum to be in the anterolateral columns and to be impaired by this operation, but it has seemed to us that when such disturbance occurs from a spinal cord lesion the crossed pyramidal tracts are damaged. We are inclined to believe that in the male impairment of sexual power possibly may result in the manner suggested. Our basis for this hypothesis is that in one case in which unilateral chordotomy was performed sexual power was lost. We mention this, feeling that one case is not sufficient to determine this matter.

The marked impairment of pain and temperature sensations after chordotomy at the fifth thoracic segment usually is pronounced to about 2 inches (5.08 cm.) above a line passing through the umbilicus, and gradually shades off above this region. We do not believe that pain sensation is conveyed in the spinal cord in association with deep pressure sensation. While pressure sensation is preserved, it is without any association with pain sensation.

Clinical observations have shown us that disease in the spinal cord is able to produce a dissociation of pain and temperature sensations,

but our observations have shown that division of the anterolateral columns produces, when successfully performed, a loss of pain, heat and cold sensations, from which we must conclude that fibers conveying these sensations are closely associated in the anterolateral column. We have observed, for example, in a case of extramedullary tumor of the midcervical region, that heat and cold sensations were greatly impaired on the opposite side of the body, while pain sensation was normal in this part. After chordotomy we have noticed that occasionally temperature sensation is lost and pain sensation greatly impaired.

Chordotomy seems peculiarly adapted to cases of malignant tumor and affords relief to the intense suffering from this affliction. It is no small satisfaction to bring comfort to one afflicted with an incurable tumor who is conscious of approaching death and is grateful for any means of making life endurable.

It is striking that there has usually been no serious implication of the motor fibers to the lower limbs. In one case (Case 3), in which the incision was more extensive because of the necessity of relieving unbearable pain, there was partial paralysis of the lower limbs. The possibility of this form of paralysis must always be borne in mind.

We have felt greatly gratified that we have had no reason to believe that extensive hemorrhage or edema of the spinal cord has resulted from the operation, although we have mentioned that in one case slight postoperative hemorrhage into the cord substance was possible. In no case was there visible hemorrhage at the site of section.

We are not surprised at the occurrence of some pain in the trunk at the level of the operation following the chordotomy and lasting a few days to a week or even longer, and on the side of the division of the anterolateral column, or even on the opposite side. This pain probably is caused by the manipulation of the spinal cord in the exposure of the anterolateral columns; it is important, therefore, that the spinal cord should be disturbed as little as possible during the operation.

We have found unilateral chordotomy sufficient for pain strictly confined to one side of the body. It is not necessary to perform bilateral chordotomy in such a case, but when the lesion is likely to implicate the opposite side of the body speedily, bilateral chordotomy is desirable, even though the pain may be unilateral at the time of operation. Some preservation of objective pain sensation does not imply necessarily that the spontaneous pain has not been removed, but it is always more satisfactory to find a complete loss of objective pain sensation in the previously painful part after chordotomy.

We have not observed trophic disturbance other than the existence of bedsores in only two of the cases, and in one of these (Case 3), microscopic examination of the spinal cord after death showed that the

crossed pyramidal tracts were seriously implicated. While there were no symptoms of pyramidal tract lesion in the other case, we believe that some injury of this tract may have resulted, even though the symptoms did not indicate this. At all events, we do not believe bed-sores are likely to develop from division of the cord confined to the anterolateral region.

REPORT OF CASES

CASE 1.—History.—J. F. C., aged 33, referred to the neurosurgical service, University Hospital, by Dr. Foster Kennedy, Feb. 9, 1922, in September, 1916, was struck in the third and fourth lumbar vertebrae by a high explosive missile and completely paralyzed from the waist down, including the sphincters. About thirty-six hours later the missile was removed. Great pain down the legs persisted. One year before admission two operations were performed on the patient elsewhere. At these operations, it was reported a number of posterior roots on the left side of the cord were cut. Following these operations, there was loss of sensation over a considerable area of the lower extremities, but the pain continued as before. Pain was always present, chiefly in the left knee and right ankle, but there were exacerbations of intense severity. It was shooting in character and at times agonizingly severe. The pain in the right ankle could be controlled by morphin, but not the pain in the left knee. At times it was even more severe in the left groin between the area of normal and of reduced sensation. Pressure and especially pin prick in this area started paroxysms of intense pain shooting down the inner side of the thigh on the left side. Occasionally it affected the left testicle. The patient had become tolerant to morphin, taking from 4 to 8 grains (0.26 to 0.52 gm.) a day. He had stopped taking it at various times during the last five years, once for a year and at other times for several months, but he had taken it continuously for nearly a year before admission. Mental vigor was unimpaired. Sexual power was present until the most recent operation, when sensation was abolished from the anterior half of the penis.

Examination.—The patient was a well developed, well nourished man. He was restless, sometimes talkative, sometimes apprehensive, and at frequent intervals stopped any occupation and cringed under excessive pain in the legs, the face becoming slightly flushed and the pulse slow. When the paroxysm had passed he talked again in a controlled voice. Extremities: The upper extremities were normal. There was atrophy and flaccid paralysis in all muscle groups in the lower extremities. There was slight power in the flexors and adductors of the right thigh, but none in the leg. The feet were in the equinus position. Patellar and Achilles' reflexes were lost. Tapping the adductor tendon produced distinct contraction on the right but not on the left.

Objective Sensation: Below the iliac crest on the left, and extending down from the buttock on the right and including the foot there was anesthesia to light touch and to pain, except for a small area over the internal malleolus which was hypersensitive. A narrow band on the right and a broad one on the left above the level of anesthesia, were hypersensitive to light touch, and especially to pin prick. On the left side, pin prick here initiated a severe spasm of pain down the left leg.

The genitalia and anus were insensitive.

Subjective Sensation: The most intense pain was felt in the left knee and the right foot along its outer edge and in the abdomen at the junction of the anesthetic and the normal sensory area especially on the left side, but in the latter place there was never pain unless the patient was examined or stimulated over this part. The inner side of the right ankle was sensitive to touch or pin prick.

Psychic effects were manifested by pain in the lower limbs. Any emotional disturbance, such as anxiety or great sympathy, caused pain in the lower limbs.

Pain occurred in either lower limb, but never in both at the same time. The pains began in the feet and surged up, on the left side, into the testicle. Pain was felt to the same level on the right but did not involve the testicle.

Bladder: By recording the amount of fluid that he had taken and the time which had elapsed, the patient knew when the bladder should be emptied, and by voluntary contraction of the abdominal muscles he could completely evacuate the bladder, especially when lying on his right side. The abdominal muscles were greatly hypertrophied and unusually strong. He had no movement of the bowels without drugs.

Operation and Course.—On Feb. 20, 1922, laminectomy and section of the anterolateral columns of the cord were performed, at the level of the fifth thoracic segment on the right side and at the level of the sixth segment on the left.

Feb. 26, 1922: The patient had little pain; the pain in his right lower limb was entirely gone, as well as most of the pain in the left lower limb.

Beginning two weeks after the operation the narcotics were gradually withdrawn without the patient's knowledge, and during the last two weeks the patient was in the hospital he was receiving no opiates.

March 16, 1922 (Dr. Spiller): Pin prick over the right thigh usually produced a sensation of pressure and occasionally of pain, but the pain sense was not normal and was delayed (that is, felt after the pressure). The same was true of the right side of the abdomen as high as the umbilicus. In the left lower limb pain sense was entirely lost and pain sensation was lost in the left side of the abdomen to about 2 inches (5.08 cm.) below the umbilicus.

Heat and cold sensations were lost over the right lower limb and the right side of the abdomen to about 1 inch (2.54 cm.) above the umbilicus, and were lost in the left side of the abdomen to about 2 inches below the umbilicus. The pain sensation was a trifle more acute over the right lower part of the abdomen than over the right thigh.

There was frequent involuntary jerking of the right lower limb, independent of any purposive irritation.

March 30, 1922: The patient was discharged, having been completely relieved of pain. All drugs were withdrawn.

April 11, 1922: A letter was received from the patient in which he said: "I really can't thank you for relieving me of so much pain, but I must make the attempt. The pain has left me in a marvellous degree."

CASE 2.—History.—G. V., aged 50, admitted to the neurosurgical service, university Hospital, June 28, 1921, referred by Dr. G. M. Piersol, presented symptoms of a spinal cord tumor at the level of the ninth thoracic segment. November 22, 1921, an exploratory laminectomy revealed a sarcoma of the vertebral column involving the spinal canal and pressing on the cord. That portion of the tumor within the canal was removed. Because of the persistent and agonizing pain, a chordotomy was advised.

Operation and Course.—Dec. 10, 1921, section of anterolateral columns was performed at the level of the fifth thoracic segment. The spinous processes and laminae designed to uncover the fifth thoracic segment were removed. The anterolateral columns on both sides were sectioned by our customary technic, that on the right side about 1 cm. higher than the left. In other particulars the operation was carried out without modification of the usual technic. A little oozing from the artery which accompanies the posterior root on the left side presented the only difficulty.

Dec. 20, 1921 (Dr. Spiller): Heat and cold were not recognized on the right side of the abdomen for 10.5 cm. above the upper margin of the umbilicus, and on the left side 7.5 cm. above the upper margin of the umbilicus. Pain sensation was lost on the right side of the abdomen to 2 cm. below the lower margin of the umbilicus; occasionally pin prick was recognized on the right side below this line in an indefinite manner. Above this line on the right side pin prick was felt, but apparently not in a normal manner until the level of the thermo-

anesthesia was approached. On the left side pin prick was not felt as high as the area of thermo-anesthesia, although occasionally it was recognized over the lower part of the left side of the abdomen in an abnormal manner. The patient was decidedly childish in her answers and tests had to be made with great care.

From the time of the operation until the patient's discharge, she was entirely relieved of the pain of which she complained before the operation. Before the operation the slightest jar of the bed caused excruciating pain; after the operation, none at all. The patient suffered somewhat from root pains at the level of the chordotomy. These have been observed after other similar operations but are only of a transitory nature.

CASE 3.—*History*.—F. S., aged 49, referred to the neurosurgical service, University Hospital, by Dr. D. D. Reynolds, Dec. 19, 1921, had her right breast amputated, April, 1919, and a resection of the left breast. The growth in the left breast recurred and she received a course of roentgen-ray treatment. In April, 1921, she began to complain of pain in the calf of the right leg and later from the knee to the ankle, chiefly on the inner side. Later the pain spread to the outer side of the leg, to a narrow zone in the back of the thigh and to the foot. It felt as though something were pressing on the sole of the foot. Later she experienced pain in the sacral region and across the buttock. November, 1921, pain began in the left lower extremity, particularly on the inner side and front of the thigh, but also in the back of the thigh, down below the knee and occasionally in the foot. She had severe pains across the lower part of the back at times and in the rectum and vagina. The pains were sharp and shooting and almost constant, although there were occasional remissions. Recently the patient suffered from pain of a neuralgic character over the left eye. She had lost little weight and felt that were the pain gone she would be well and able to take care of her house.

Dec. 23, 1921: Both patellar reflexes were very prompt, but equal and a little exaggerated. No Achilles' reflex was obtained on the right side. A normal Achilles' reflex was present on the left. Laségue's sign was distinct on the right; it was slight on the left. Pain was present in both hip joints, not so severe in the left, and it followed the sciatic nerve on the right; but there was more in the front of the thigh than on the left. It was painful to sit on the right buttock. No bladder or bowel disturbance was present. She was never without a sensation of discomfort, and pain was severe every day, especially toward evening. Moderate degrees of heat and cold were promptly and correctly recognized in all parts of the lower limbs and over the whole of the abdomen and chest. The point was always recognized correctly from the head of the pin in the same parts. At times she did not seem to have acute pain sensation over the abdomen, but there was probably no real loss of pain sensation.

Roentgen-Ray Report: There was a metastatic carcinoma of the fifth lumbar vertebra and both iliac bones, especially on the right side, and probably of the upper part of the right femur. Chest: There was a mediastinal shadow which was suspicious; the right diaphragm was very high, suggesting metastatic growth in the liver.

Dec. 21, 1922: Bilateral section of the anterolateral columns was performed. Part of the first, all of the second, third, and part of the fourth thoracic spinous processes and laminae were removed. This gave ample exposure to the fourth and fifth thoracic segments. We were a little uncertain at the time as to whether we had exposed the fourth and fifth or the fifth and sixth thoracic segments, as two pairs of roots seemed to come off opposite the base of the third thoracic vertebra, one opposite the upper portion and the other opposite the lower portion, and it was at these respective levels at a distance of 1 and 0.7 cm. apart, that the sections were made, the higher section on the right, the lower section on the left. Not a drop of blood escaped within the spinal canal during the operation. Section was made as far as

could be determined by observation, 3 mm. deep and broad, the knife penetrating at a point midway between the posterior and anterior roots and coming out at the anterior roots.

Dec. 23, 1921: Heat and cold sensations began to be clearly recognized on the abdomen about 1 inch above the upper level of the umbilicus on the right side. The area on the left side was not determined because the patient was too tired. There was no clear perception of temperature sensation in either lower limb. Occasionally, she answered correctly for temperature over the lower part of the abdomen on either side. It is uncertain whether this was a guess or not. Pin prick caused no pain below the knee on either side. On the front and inner side of each thigh she recognized pin prick occasionally, but it did not seem to produce the usual pain. She rotated the left lower limb at the hip, had slight flexion of the toes of her left foot but no extension of the toes and no power at the ankle. The same was true of the right lower limb, but the voluntary power was not quite as great in the left lower limb.

Dec. 31, 1921 (Dr. Spiller): There was no distinct recognition of pin prick over the front and inner side of either thigh, although she answered correctly occasionally concerning pin prick of these parts. Pin prick was felt indistinctly over the lower part of the abdomen on either side, but it required a fraction of a minute before she was able to form an opinion of the irritation, and the pin prick was not as sharp as it was at higher levels where it was recognized instantly. This hypalgesic area extended to about $1\frac{1}{2}$ inches (3.81 cm.) above the upper level of the umbilicus on the right side and a little higher on the left side. It shaded off gradually into the normal area. She had not had any pain in her lower limbs since the operation. She said that before the operation she "suffered tortures," and that her condition after the operation was "like heaven," and that it was "marvelous." She had had three months of terrible suffering, waking on account of the pain after dozing a short time, even after morphin. She was most enthusiastic in her expressions about the relief of pain.

Since the operation she had had incontinence of feces. For nine days she had had retention of urine and was catheterized every twelfth hour. On December 31 she had incontinence of urine.

Jan. 5, 1922: Power in the lower limbs was much greater than immediately after the operation.

Jan. 18, 1922 (Dr. Spiller): She was able to flex the leg on the thigh and draw the foot up on both sides. This was done with effort. There was difficulty in dorsiflexion of the foot and toes.

Tactile sensation was normal everywhere about the lower extremities.

Heat and cold sensations were not entirely lost anywhere in the lower limbs, but were greatly disturbed in the lower limbs and over the abdomen. Extremes of temperature were occasionally correctly recognized in the lower limbs and still better over the abdomen.

There was great disturbance of temperature sensation to a level about 3 cm. above the umbilicus on the right side and somewhat higher on the left side. There was not a sharp line of definition.

Pain sensation was affected in about the same manner as temperature sensation. A deep pin prick was occasionally recognized as sharp over the lower limbs and still more so over the abdomen to about the same height as the disturbed temperature sensation on the right side and about 7 cm. above the umbilicus on the left.

The shading off of the area of anesthesia for pain and temperature was so gradual over the abdomen that it was impossible to make any sharp definition.

She could draw up either lower limb at the hip or knee and extend it. She could also flex the toes of each foot, but had no power in dorsiflexion of the ankle or the toes.

The patellar reflex was very feeble; indeed it apparently was absent on both sides even with reinforcement. The Achilles reflex was absent on both sides. The lower limbs were distinctly flaccid.

She still had a little pain between the shoulders in the region of the operation, but since the operation she had had no pain in the lower limbs or in the abdomen. She had had a good deal of distention of the abdomen, and when this had occurred she had had a great deal of discomfort. This was not like the pain from which she had suffered previously.

She was most enthusiastic over the relief from the intense pain she had experienced before operation.

Jan. 19, 1922: The patient was discharged.

A letter received from the patient's husband, dated March 20, 1922, stated that the operation had caused complete cessation of the pain in the lower limbs. The operation, therefore, was entirely successful in the removal of the pain for which it was performed. The patient had to be catheterized.

Necropsy Examination.—Removal of the spinal cord by Dr. Grant was permitted after death. Carcinoma nodules were found on the roots of the cauda equina. A transverse section of the cord at the fourth thoracic level, stained by the Marchi method, showed considerable degeneration of the peripheral portion of the anterolateral columns and of the anterior portion of the direct cerebellar tracts. The portion of the white matter near the anterior horns did not show recent degeneration, and evidently these fibers do not degenerate upward. The section was above the area of operation. These fibers were degenerated below this level. A section made at the sixth thoracic level showed considerable degeneration of the crossed pyramidal tracts and of the white matter adjoining the anterior horns, even in the anterior columns, but not much of the periphery of the vertebral columns.

CASE 4.—History.—Mrs. W. W. K., aged 34, referred to the neurosurgical service of the University Hospital by Dr. A. M. Ornsteen and Dr. William G. Spiller, Nov. 19, 1921, was well until five years before, when she began to void at frequent intervals, with pain during micturition. About one and one-half years after the onset she began to have pain in the left side of the vagina and rectum, also in the sole of her left foot. These pains were shooting in character, and came on at intervals of a few days and lasted for several days. During some of these attacks, she would have to stay in bed, and walking would make the pain worse. The condition became worse, and in the summer of 1920 she was operated on by Dr. Floyd Keene, who removed two Hunner's ulcers from the bladder. She had been slightly better since. She voided every hour during the day and night and had considerable pain doing so. Lying on her back gave her pain in the left sacral region. The pain was controlled only by morphin. She took almost 2 grains (0.13 gm.) a day.

Examination.—Deep reflexes in the upper extremities were normal. Patellar reflexes were exaggerated and equal. Ankle reflexes were exaggerated, the left more so than the right. Sensation was practically normal, except for a slight hypesthesia and hypalgesia in the fifth sacral segment (anal region).

Operation and Course.—Nov. 26, 1921: Section of the anterolateral column at the level of the fifth thoracic segment was performed.

The spinous processes and laminae of the fourth and fifth thoracic vertebrae were removed so as to expose the fifth thoracic segment. When the dura was opened, that portion of the cord, including the right anterolateral column, was grasped with the needle and section made with a knife, 2.5 mm. deep. The knife was plunged straight in so that the section was square. At first it appeared as though the section might have been too deep, at least the diameter of the cord at this level was quite small and the needle looked relatively large.

Nov. 29, 1921 (Dr. Spiller): The patient had pain in the right side of the vagina and rectum for twenty-four hours after the operation. On November 29, she complained of pain in the lower part of the right side of the abdomen. She had pain over the right side, partly as a result of the operation. The right lower limb was a little paretic. The patellar reflex on the right side was a little prompter than normal; she had right abortive ankle clonus and a typical Babinski reflex, but moved the entire right lower limb freely, even the toes.

For twenty-four hours after the operation she did not move the right lower limb. She had complete loss of heat and cold sensations over the whole left lower limb and abdomen up to 5 inches (12.7 cm.) above the umbilicus and extending close, but not fully to, the median line. Touch sensation was normal on the left side.

Dec. 1, 1921 (Dr. Spiller): The loss of pain, heat and cold sensation was complete on the left side to about 2 inches (5.08 cm.) above the umbilicus. The pain on the left side had stopped completely, but she complained much of pain over the right side of the trunk extending into the right side of the vagina; the pain was worse in the lower part of the right side of the abdomen. This pain may have been caused by the operation and referred from the site of operation, or it may have been caused by lying on the abdomen during the operation. The impairment of motion in the right lower limb had almost disappeared; the right knee reflex was a little exaggerated, but there was no ankle clonus on the right, although there was a typical right Babinski reflex. She moved the entire right lower limb and toes freely. The loss of pain and temperature sensations on the left side of the abdomen was well defined. She had no diminution of pain and temperature sensations in the right lower limb or abdomen.

Dec. 3, 1921 (Dr. Spiller): All pain in the lower part of the right side of the abdomen stopped December 2, and on December 3 she had full control over the right lower limb.

Dec. 10, 1921 (Dr. Spiller): The patient left the hospital on this date. She limped a little on the right, but the old pain had not been present since the operation. Movement in the lower limbs was normal, except that she dragged the right lower limb a little as she walked. There was no change in the reflexes.

March 4, 1922: The pain for which the operation was performed had not returned.

Sept. 14, 1922: Pain had persisted in the right side of the vagina, right side of the bladder, outer side of the right thumb, and heel and posterior half of the outer border of the right foot, and chordotomy of the left anterolateral was necessary. The operation had removed all pain from the left side of the body, and it was performed to accomplish this. She had a little subjective weakness of the right lower limb, felt only when she tried to hurry in walking.

CASE 5.—History.—Mrs. H. W., aged 37, referred to the neurosurgical service of the University Hospital by Dr. Curtis Burnam and Dr. R. M. Lewis, April 22, 1921, in August, 1917, had had a colostomy performed for rectal carcinoma. December, 1917, radium therapy had been administered under the direction of Dr. Howard Kelly. In January, 1918, a modified Kraske operation had been performed by Dr. Kelly. There was general improvement until May, 1919. From that time to April, 1920, her condition became worse, and she began to use opiates. Since then the pain had become more and more unbearable, and she had resorted to increasing doses of opiates. The pain was referred to the sacrum and down the right leg, sometimes to both legs. The pain exceeded her endurance and she found life unbearable. Before operation she required 3 grains (0.19 gm.) of morphin daily.

Examination.—The patient was emaciated and appeared cachectic. The lower extremities were markedly atrophied, and the patellar reflexes exaggerated.

Operation and Course.—April 30, 1921: Section of the anterolateral columns, right and left, at the level of the tenth thoracic segment was performed. The spinous processes of the ninth, tenth and eleventh and the laminae of the tenth and eleventh thoracic segments were removed. The cord was rotated first in one direction, then in the other, and with the special needles the anterolateral columns were isolated and sectioned.

The day after the operation the patient complained of a sensation of constriction as by a tight band about her waist at the lower margin of the ribs. At times for several days she had a sense of suffocation, which was difficult to explain.

The wound healed by first intention, and the stitches were removed on the sixth day. On the eighth day, the patient was up in a wheel chair. Ten days after the operation all morphin had been withdrawn, and the patient was free from pain. She could not appreciate the fact that her pain had been entirely subdued.

Eleven days after operation, the patient became drowsy, and on the thirteenth day she died of meningitis. The relief from pain in this case was striking. The development of a delayed infection almost two weeks after the operation was hard to explain, except on the grounds that in the cancerous cachexia of four years' duration the patient's resistance had been lowered. Subsequently, she was free from pain, but no objective examination had been made as to loss of pain and temperature sense.

CASE 6.—History.—A. B., aged 53 years, was admitted to the neurosurgical clinic of the University Hospital, Oct. 28, 1921. In August, 1919, a carcinoma of the cecum was removed. There were no adhesions and apparently no enlarged glands. The patient made a good recovery, and was well until February, 1921, when she began to have pain and paresthesia in the distribution of the right external cutaneous nerve—much like meralgia paraesthetica—and soon anesthesia and analgesia. A second exploration in April, 1921, revealed an inoperable mass.

The pain was constant most of the time, severe, aggravated by walking, and was wearing out the patient. Morphin was not tolerated; it caused nausea and vomiting. If the patient were free from pain, life might be prolonged a considerable time in comparative comfort, as there was no evidence of intestinal obstruction. With this understanding and with this in mind, the patient was referred by her physician to Dr. Frazier for a chordotomy.

Operation and Course.—Oct. 31, 1921: A chordotomy of the left anterolateral column was performed. The laminae and spinous processes of the fourth and fifth thoracic vertebrae were removed. By traction on the dentate ligament, the cord was slightly rotated. The special curved needle was introduced at a point midway between the posterior and anterior roots, and made its exit at the anterior root. The cord tissue included in the curve of the needle, representing a section 2.5 mm. broad and 2.5 mm. deep, was divided with a cataract knife. There was no bleeding.

Nov. 12, 1921: Tactile sensation was normal in the right lower extremity, except in the former area of anesthesia. Pain sensation was not felt distinctly as pain, but was felt sufficiently to permit recognition of the point from the head of the pin. This sensation was at times one of pricking from the point of the pin or of scratching by the fingernail, but was less acute than that of a pin prick on the other limb; it was readily exhausted. While fairly acute at the beginning, the recognition of the pin point was lost for most pricks made later. This disturbance of pain sense was present in the whole right lower limb and also, though less marked, over the right side of the abdomen about up to the level of the umbilicus. Over the right side of the abdomen pain sensation was less affected than in the right lower limb and also less affected about the level of the umbilicus. In this right abdominal area, pin prick was usually recognized, but much less acutely than on the left side. On the left side the plantar reflex was normal; ankle and knee reflexes on the left were a trifle more prompt than normal; possibly there was an abortive ankle clonus on the left. Thermal: Ice and hot water were not recognized on the entire right lower extremity. All over the lower part of the right side of the abdomen ice was called pin prick to 2 or 3 inches above the umbilicus, and very hot water was occasionally recognized correctly, but with much less intensity than over the corresponding area on the left side.

Dec. 12, 1921: Pain, heat and cold sensations were lost in the right lower limb, immediately recognized on the right side of the abdomen above Poupart's ligament, but not so keenly, and these sensations became more acute as the region of the umbilicus was approached. On the right side of the abdomen

she confused heat and cold sensations. She could not distinguish one from the other clearly to about 3 inches above the umbilicus. While she recognized pin prick on the right side of the abdomen, the sensation was not as sharp as on the left side of the abdomen. The impairment of temperature sensation extended a little higher than impairment of pain sensation on the right side of the abdomen. There was no weakness of the left lower limb, and no upward movement of the left big toe in the Babinski reflex. There were no diminution of pain, heat and cold sensations in the left lower limb. She had "creeping sensation," that is, some numbness from the right hip to the knee.

She had had almost entire relief from pain on the right side since the operation.

She had had a sense of fatigue in the left side of the abdomen and the left hip since the operation; this was not a true pain, but more in the nature of an ache, and was sometimes relieved by changing the position.

Feb. 19, 1922: There was an indefinite area about the inner and anterior aspect of the knee, in which a needle prick was distinctly perceived as sharp, and was decidedly uncomfortable. The analgesia extended to about the inguinal region, but the horizontal upper border was not sharp. Above that, as high as the level of the umbilicus, there was an area in which pain sense seemed rather less acute on the right than on the left, but testing with a sharp needle showed there that pricks were painful. There had not been a return of spontaneous pain in the right lower limb. Indeed, there had not been severe pain anywhere.

CASE 7.—History.—H. E. H. D., aged 47, admitted to the neurosurgical service of the University Hospital, Aug. 26, 1921, on Oct. 21, 1916, was wounded by a high explosive shell in the battle of the Somme. A diagnosis was made of compound fracture of the right ilium with sciatic nerve involvement. For four weeks following the operation the right leg was paralyzed and numb. He had had no sensation from the hip down. When sensation and motion began to return, the pain developed, soon becoming severe. He remained in army hospitals for eighteen months, the wound in the back healing one month before discharge. He returned to his home in Canada in May, 1918, suffering severe pain without any relief from medication. In May, 1919, he was operated on at the University Hospital (chordotomy), and for six weeks subsequent to operation he was free from pain, though conscious of a sensation in the foot, which was not annoying. The "real pain" returned about the end of July or August, 1919. The pain before the operation was referred to the thigh as well as the foot; in fact, it was more intense in the thigh than at any other place. Since the operation, when the pain returned, it was referred only to the foot and had continued there ever since; it began in the heel and extended along the sole to the big toe. The pain varied in intensity, but in some degree it was continuous, almost always confined to the foot, but occasionally felt in the lower part of the thigh, posteriorly.

Examination.—Aug. 31, 1921 (Dr. Spiller): Sensation: There was a distinct diminution of heat, cold and pain sensations in the right side of the abdomen below the level of the umbilicus, still greater in the anterior part of the right thigh and still greater on the inner side of the right leg. This impairment was present to about 1 inch above the umbilicus, but shaded off so gradually into the normal sensory area that a sharp definition was impossible. Tactile sensation was normal over the right side of the abdomen, anterior part of the right thigh and inner side of the right leg. It was greatly impaired down the outer side of the leg and dorsum and sole of the right foot.

The patient said that the pain was precisely as it was before the operation in both frequency and severity. It was evident that he had had a partial but not a complete interruption of the pain and temperature fibers from the right lower limb.

Reflexes: The left Achilles reflex and the left patellar reflex were normal. The right patellar reflex was weak. The right Achilles reflex was absent.

Operation and Course.—Sept. 20, 1921: Two spinous processes were exposed and isolated above the previous laminectomy, but only one of these, the lower one, was removed, together with the corresponding lamina. There had evidently been partial regeneration of the lamina next below. The special needle was introduced at a point on the left aspect of the cord midway between the origin of the anterior and posterior roots. As much tissue as the needle would contain was divided. There was no bleeding within the dural sac, with the exception of that which arose from the artery accompanying the posterior root at the level of section.

Sept. 21, 1921 (Dr. Spiller): On September 20, an attempt was made to cut one segment higher than at the previous chordotomy on the left side. As a result, the patient had complete loss of pain, heat and cold sensations in the anterior part of the right thigh and over the right side of the abdomen up to a line passing about 1 inch above the umbilicus. Here the differentiation between pain and cold was sharp, but for heat not quite so sharp. This level corresponds to the eighth thoracic segment. The patient had attacks of pain on the evening of the operation beginning in the right foot and not extending above the ankle. There was a sensation of constriction quite unlike anything he had ever had before, possibly the result of irritation of the central end of the fibers in the spinal cord. This sensation of painful constriction was not associated with any sensation of burning or coldness. The left lower limb felt heavy, but he moved it freely and wiggled the toes.

Tactile sensation was preserved in the anterior part of the right thigh and in the right side of the abdomen.

Sept. 26, 1921 (Dr. Spiller): On September 24, the patient had paresthesia in his left foot, which persisted, although it was not continuous. He could not describe the condition satisfactorily. The condition was not unlike the sensation produced by an insect crawling over the foot, but was not always of the same character. It was never painful. He still had complete loss of pain sensation to about 1 inch above the umbilicus on the right side. The pain, which had developed since the operation, persisted in the right foot; it was unlike any sensation he had ever had previously, but he said that if he never had any more acute pain, he could bear it easily. The pain about the chest, in a band just above the level of the operation, was probably due to the manipulation of the cord at the operation, and was slight.

Sept. 28, 1921 (Dr. Spiller): Paresthesia of the left lower limb had been less during the last twelve hours than at any time since September 23. This sensation had never been one of pain. The patient was entirely unable to describe it adequately. The sensation was not one of numbness, tingling, the feeling of insects crawling over the skin, or of the part "being asleep." The sensation of pain for pin prick and of heat and cold, objectively tested, was normal in the left foot, as in the left upper limb. Pain, heat and cold sensations were still completely lost to about 1 inch above the umbilicus on the right side. Spontaneous pain in the right foot had been less during the last twelve hours.

Oct. 5, 1921 (Dr. Spiller): The curious feeling of paresthesia in the left foot was much diminished, but the pain in the right continued practically unchanged.

Oct. 8, 1921 (Dr. Spiller): Heat and cold sensations were completely lost in the whole right lower limb and over the right side of the abdomen as high as a line about 1 inch above the umbilicus. Above this for about 2 inches there was a disturbance, but not loss of heat and cold sensations, on the right side only. This was true also of pain sensation. He did not feel a pin prick in the whole right lower limb and as high as a line about 1 inch above the umbilicus. Above this the sensation to pain was distinctly diminished for about 2 inches.

There was not the slightest impairment of motor power in the left lower limb, nor had there been any since the operation. Tactile sensation in the sciatic distribution of the right side was the same before the operation. The

right side of the scrotum had normal tactile sensation. The left side had normal sensation. The right side of the penis had diminished, not lost, temperature and pain sensations and normal tactile sensation. The left side of the penis had normal sensation. Sense of position was normal in the left big toe. The right big toe was affected by the old injury. The right patellar reflex was normal; the left was distinctly exaggerated, but there was no patellar clonus. There was persistent ankle clonus on the left side. The Babinski reflex was uncertain on the left side. There was no distinct movement on either side, although the movement seemed to be downward.

The patient had been walking frequently and had noticed no weakness in the left lower limb. He still had the same pain in the right foot that he had immediately after the operation. It had been of about the same intensity and frequency, possibly a little more frequent during the last week; it was very different from the pain he had had before the operation. It was not nearly as severe as the pain before the operation, but the frequency was about the same. A deep pin prick in the analgesic area was not perceived as pain.

Oct. 9, 1921: The patient was discharged.

Second Admission.—Feb. 20, 1922 (Dr. Spiller): The patient was readmitted to the hospital. After leaving the hospital following the second operation (chordotomy, September, 1921), the pain in the right foot gradually increased and finally involved the whole of the sole of the foot but not the dorsum. There was no spontaneous pain in the dorsum of the foot.

Until about three weeks before he had had no other pain; then he began to have pain about the middle of the posterior surface of the right thigh over the course of the sciatic nerve. The pain did not occur in the thigh unless he had pain in the right foot, and was present in the right thigh only when the pain in the right foot was intense; it seemed to radiate upward from the foot. He had some pain in the right foot all the time, but it was usually of a mild character, with an occasional exacerbation, severe enough to cause him great discomfort, but not as severe as the pain before the first chordotomy. While his sleep was disturbed by pain, he did not take drugs to control it; he admitted that his pain was decidedly less than before the first chordotomy.

Feb. 23, 1922 (Dr. Spiller): Over the dorsum of the foot and the sole on the right side all forms of sensation (touch, pain, heat and cold sensations) were lost. On the outer side of the right leg and down the posterior side of the right thigh, touch sensation was very indistinct, that is, throughout the distribution of the right sciatic nerve. Elsewhere in the right lower limb, and over the front of the right lower part of the abdomen, tactile sensation was evidently normal.

Heat and cold of extreme degrees (ice and very hot water) were not recognized anywhere in the right lower limb or over the right side of the scrotum, or on the right side of the abdomen to about 1 inch above the level of a line passing through the umbilicus. Pin prick was felt in the right lower limb only in an area on the inner side of the right leg, beginning quite close to the patella, bounded on one side by the crest of the tibia and on the other by a parallel line 1 inch to the inner side. This area widened as it extended to the heel, and terminated at the beginning of the sole of the foot. In the lower part of this area of preserved pain sensation, slight percussion with the finger caused a stab of pain. Deep pin prick anywhere else in the right lower limb, including any part of the thigh and the right side of the lower part of the abdomen, as high as the umbilicus, was not appreciated.

After sitting for some time there was momentary weakness in the left lower limb. No weakness of the left lower limb could be detected in resisting active movement to it. There was no ankle clonus on the left side. The left Achilles reflex was normal. The left patellar reflex was not unusually prompt, but it was a trifle more so than the right.

It would seem from this examination that the pain fibers from the lower and inner part of the right leg had escaped in the chordotomy, and this would

indicate that they were probably the more posterior or interior fibers of the anterolateral tract of the left side.

Second Operation and Course.—Feb. 24, 1922: A resection 6 cm. long was made of the saphenous nerve under local anesthesia. The nerve was exposed by a vertical incision after it had divided into its two terminal branches. Pinching one of these elicited pain precisely in the region in which the patient had been complaining of pain so bitterly, that is, in the inner side of heel. The branches were traced up to the main trunk where the upper section was made. After the resection, pinching the central stump elicited pain on the inner side of the foot and heel.

Feb. 26, 1922: The patient said that this was the first time in five years that he had been entirely free from pain or disagreeable sensations of any kind, except when under the influence of drugs. Before, when the acute pains were not present, a peculiar sensation had annoyed him. He could hardly believe that it was true, but he had had no pain or sensation in his foot for more than two hours on this date. His wife said that, to her knowledge, this was the first time in five years that he had been free from pain.

Feb. 27, 1922 (Dr. Spiller): When seen, on February 24, he was having severe pain in the toes of the right foot, probably as severe as before the operation, but possibly not as frequent. He had had a good night following the day of operation, and when seen the next morning (February 25) he was still suffering a great deal of pain in the toes of the right foot. He suffered great discomfort in the right foot all day.

He had had much better nights since February 25, and since then had felt remarkably better. He had practically no pain all day on February 26, having intervals as long as two hours without the slightest twinge of pain or sensation of any sort in the right lower limb. After an hour or two he might have a twinge of pain with drawing up of the right lower limb. It was never severe and of only a few minutes' duration. He went to sleep the evening of February 26 "like a child," the first time since the injury.

On February 27, there was slight pain about the middle of the side of the right foot. On February 25, the intense pain of the preceding night was believed to have been caused by the irritation of the central stump of the long saphenous nerve after section, the nerve not yet having lost its function. He had never felt so encouraged before.

March 7, 1922 (Dr. Spiller): The patient said that the pain was over the whole of the sole of the right foot, but that it was more intense over the inner half than over the outer half.

Third Operation and Course.—March 11, 1922: A laminectomy and chordotomy was performed on the left. Section was made, 3 cm. deep, at the level of the tenth thoracic segment.

March 12, 1922 (Dr. Spiller): Touch, pin prick, heat and cold apparently were normally recognized throughout the whole left lower limb. He did not move the left toes freely on account of suffering from the operation of the previous day. No further examination was made. The day and evening of the operation, he had some pain in the right toes, which continued. It was located chiefly in the right toes. Before the operation it seemed to be chiefly in the sole.

March 14, 1922: The patient had his first bed bath on the morning of this date. While being washed on the outside of the left thigh, he had a peculiar sensation similar to that which he had had for years in his right hip. It was almost a numb sensation. He felt the pin prick as distinctly in the right as in the left thigh. Occasionally he seemed to feel the pin prick a little less acutely in the left thigh.

Heat and cold sensation, however, were distinctly impaired in the left thigh. He repeatedly called the ice cold tube "hot" at any part over the thigh, although sensation was a little more acute over the posterior surface. He

could not readily appreciate heat in the same region. Below the left knee, temperature sensation, though impaired, was not lost.

The patellar and Achilles reflexes on the left side were normal. There was no clonus on the left side. The right patellar reflex was absent. The Babinski test gave no distinct flexion of the left toe. There was no weakness of the left lower limb.

The patient had a little less pain during the morning of March 14 than before. It came in paroxysms.

March 16, 1922: The sensation of pin prick in the left thigh was fully as sharp as in the left upper limb. The same was true of tactile sensation. During the previous night and the morning of March 16 he had "scarcely any pain." Heat and cold sensations were practically lost over the front and on each side of the left thigh, but only a little, if at all, impaired, over the back of the thigh.

Sensation for heat and cold was greatly impaired over each side of the abdomen as high as the nipples (that is, the fourth intercostal space). Pin prick sensation was lost to about 3 inches (7.62 cm.) above the umbilicus. The loss of heat and cold sensation on the left side of the trunk up to the fourth interspace suggested temporary injury of the right side of the cord sustained during the operation.

March 20, 1922 (Dr. Spiller): The patient complained of considerable "burning" pain in the whole sole of the right foot. It was, therefore, associated with a sensation of heat. He said that he had been subject to muscle cramps all his life, but this pain was not of the same character. All the pressure made with the hand in grasping the limb produced no pain in the right thigh or foot. The spontaneous pain, therefore, apparently was not in the deep peripheral sensation.

March 22, 1922: His tactile sensation and pressure sensation were acute in the right thigh, but all the pressure that could be brought to bear with a fountain pen over the femur of the right thigh produced a distinct sensation of pressure, but not the slightest discomfort; whereas a moderate amount of pressure over the left thigh produced discomfort, and then pain. Vibratory sense was normal and equal as tested over each patella.

March 28, 1922 (Dr. Spiller): Sensation to touch and pin prick was normal in the thigh and leg on the left side and was equal in intensity with the sensation of the left upper limb. There was a distinct impairment in heat and cold sensations in the left lower limb and left side of the abdomen to a level of 2 inches below the nipple. In this area, ice-water was repeatedly called "hot." Very hot water in this area was always recognized correctly as "hot." Careful testing of pain sensation in the left thigh and leg failed to elicit any disturbance of pain sensation. The peculiar paresthesia which he had noted in the left thigh previously when being bathed, had entirely disappeared.

March 29, 1922 (Dr. Spiller): The patient's wife said that after the first operation, May 24, 1919, he had had no pain of any consequence for three months, and every operation had been followed by relief of pain for an average of three months, although the relief had not been absolute. After the second spinal operation a trip to Florida caused great improvement in his condition. He had less pain while there. When he was in the company of strangers, he seldom showed pain. There evidently was a psychic element in his pain. He was much more likely to have pain when alone with his wife than when with others, at which time, if any argument arose, the pain immediately increased.

March 31, 1922 (Dr. Spiller): His nurse said that for four nights and three days the only medicine he had received for relief of pain was a capsule of sugar, and this had always given relief. He had asked for it again when pain had recurred. The only drug he had received to make him sleep during the two previous nights had been a capsule of sodium bicarbonate, and he had slept as well after taking this as he did after taking barbitol. This statement may be offered as evidence that his pain had a large psychic element.

April 1, 1922: The patient was discharged.

June 12, 1922: The patient reported that the pain was still present in the sole of the foot; it was only occasionally present in the lower part of the thigh and was not as severe as formerly. There was no abnormal sensation in the "good" lower limb; but when he awakened in the morning, or after sitting for some time, he could not use this limb, possibly for half a minute.

CASE 8.—*History.*—E. S. S., Jr., aged 40 years, referred to the neurosurgical service of the University Hospital by Dr. Hugh T. Patrick and Dr. T. J. McKinney, said that he had been entirely well until 1 p. m., May 12, 1921. While attending a committee meeting, he began suddenly to have severe pains in the back about the lumbar region. The pains came in waves, and at first he thought them due to his bowels. An hour later the pains became so severe that he became pale and weak. He went to a physician's office. The physician thought there was some intestinal disturbance, and gave him a drastic purge. He said he told the physician that he felt as though he had lumbago, and he rested in the physician's office for a time. Later he walked home with difficulty, and neighbors told him that his limbs seemed to drag. He noticed that his legs seemed to be going to sleep, and he was unable to use them. When his wife returned she assisted him to bed; he was able to lift the left leg up on the bed, but not the right. The next day he felt a definite girdle sensation around the waist, about at the level of the umbilicus. About two weeks later he had another attack, which consisted of severe pains in the back, radiating around to the sternum just below the nipple line. Later the girdle sensation disappeared, and he gradually lost sensation up to the nipple line. The second attack seemed to involve the neck and arms. He was unable to use the arms for about two weeks. The eyes required subdued light. The level of the lesion had remained the same as that after the second attack, and he had had no further "attacks." There had been a constant pain, dull in character, in the right side and in the back. During the first attack, there was some febrile reaction, which continued through the second attack.

Since the second attack there had been a band of hyperesthesia about the chest. This was so painful in the right axilla that it was necessary to prevent contact over this area. The bed clothing was not bearable, and he held it off with a frame or with his arm. Since the first attack he had lost all motor and sensory function below the waist. He had been incontinent in regard to both urine and feces. During the last two months, he had had five attacks of hematuria, lasting from three to seven days.

His brother, a physician, said that he and the patient were "bleeders," and that slight cuts caused quite severe hemorrhages. The patient asserted that he had had no serious hemorrhages, except after the extraction of a wisdom tooth.

Physical Examination.—Head, neck, heart and lungs were normal. Decubitus was present over the buttocks on each side, and there was one sore above the coccyx.

Lower Extremities: There was absolute flaccid paralysis of both lower extremities. There was loss of tactile pain and temperature sense below the level of the sixth rib.

June 5, 1922 (Examination by Dr. Spiller): The patient's wife said that for two months after the onset of his symptoms he had had no hyperalgesia. Hyperalgesia began about the end of August, 1921. The condition of the lower limbs was one of complete flaccid paralysis, with much atrophy and complete loss of sensation for touch, pain and probably all other qualities. He complained of a burning sensation in the right knee; he felt this sensation occasionally in the left knee. The pain about the chest was much more severe on the right side, and extended from the spinal column all the way to the midline of the chest. The pain was in a band about 2 inches wide; the upper border of which was at the level of the nipple. The pain in the left side was

only in front and extended about 1 inch above the nipple. It was stinging, burning or throbbing, always present, sometimes with severe exacerbations, and worse at night. The zone of irritability to touch was wider than the zone of spontaneous pain. The zone of hyperirritability to touch began on the right side at about the third intercostal space, and extended to about 2 inches below the right nipple. It was about the same in width over the left side of the chest. When this zone was touched, the patient winced and showed pain; even touching the hair in this area caused a stinging sensation—not sharp pain. There was no increased sensitiveness to touch or pin prick in either axilla. The pupils were equal and normal in size. There was no widening of the palpebral fissures and no protrusion of the eyeballs, that is, no evidence of ophthalmic sympathetic involvement. The patient said that the hypersensitiveness of the chest had been getting worse during the last three or four months.

RESULT OF TESTS OF REFLEXES

Reflexes	Right	Left
Biceps	++	++
Triceps	++	++
Abdominal	0	0
Patellar	0	0
Achilles	0	0
Babinski	0	0
Clonus	0	0

Urinalysis was negative. Blood examination revealed that the clotting time was six and one-half minutes. The spinal fluid pressure was 10 mm. of mercury, and the spinal fluid contained four cells; it was negative for globulin and sugar. The Wassermann blood and spinal fluid tests were negative. Roentgenologic examination was negative.

Operation and Course.—June 7, 1922: An anterolateral section, bilateral, at the level of the first thoracic segment was made. Beginning under local anesthesia, the spinous processes and laminae of the seventh cervical and the first thoracic vertebrae were removed. The oozing from the margins of the laminae and from the muscle surface was unusually stubborn, and continued so throughout the operation. Therefore, the field was not nearly so dry as we would have wanted to have it before the dura was opened. When the dura was opened it was found that a posterior root on the left side, presumably either the last cervical or the first thoracic, was given off exactly in the middle of our laminectomy opening and just opposite our guide suture, which corresponded to the tip of the seventh cervical vertebra. As the laminectomy opening was small, it was rather inconvenient to have the posterior root given off exactly in the middle of the opening, but with a silk suture it was gently retracted upward, and the root section was made immediately below. Right Side: The root on the right side came off at a slightly lower level. It was retracted gently downward, and the chordotomy section was made just above it. It was noted that throughout the operation when the patient complained of pain it was referred to the arms. The dural incision was closed and considerable time was spent in trying to control the oozing from the various points in the wound; after one point was closed another would begin to ooze, so that we became rather discouraged, although with the aid of muscle grafts and warm tampons we were finally able to secure a sufficiently satisfactory hemostasis to warrant our closing the wound. This was done with interrupted tier sutures of silk. A Mickulicz drain was introduced in the lower angle of the wound. Convalescence was uneventful. The wound healed by first intention.

June 8, 1922 (Examination by Dr. Spiller): Both pupils were very small, the right smaller than the left. The palpebral fissures did not seem to be as wide as normal, and the right was not quite as wide as the left. He had spontaneous pain in the hypothenar eminence of the right hand, and while pin prick was felt here as sharp, it was not quite as acute as it was over the right thumb. He had, therefore, symptoms indicative of ophthalmic sympathetic involvement, more on the right than on the left. Morphia may have been partly responsible for the contracted pupils. It hardly seemed to explain the whole finding because of inequality of the symptoms on the two sides and because of the spontaneous pain and slight hypalgesia in the distribution of the first thoracic nerve. The grasp in the right little finger did not appear to be quite as powerful as the grasp in the left little finger.

June 9, 1922 (Examination by Dr. Spiller): In looking straight forward without making any special effort, the palpebral fissures were distinctly subnormal in width, the right more so than the left. In making an effort to open the eyes wide the fissures were wider, but still subnormal. The right pupil was a little smaller than the left. The patient had complained during the past twenty-four hours of considerable pain and numbness down the inner side of each forearm and in the ring and little finger of each hand, more in the right than in the left. The spontaneous pain and hyperalgesia to pin prick over the upper part of the thorax had disappeared entirely.

June 27, 1922 (Examination by Dr. Spiller): The right pupil and the right palpebral fissure were distinctly smaller than the left, and the right eyeball did not protrude as much as the left. When asked to open his eyes, or to look upward, the palpebral fissures were about normal. The reaction of each pupil to light and in convergence was normal. The flexor power of each little finger was still very weak. The pain that was present after the operation on the ulnar side of each forearm and hand had entirely disappeared, but he still complained of numbness—of less intensity than immediately after the operation—in the little and ring fingers of each hand and extending up the ulnar side of each forearm and corresponding part of each arm as high as the axilla. There was diminution in touch, pain, heat and cold sensations, but no loss on the ulnar side of the left forearm, in the corresponding part of the left arm above the elbow or in the ulnar side of the left hand. This disturbance of the same character was greater in the right upper limb than in the left and was in the ulnar side of the right forearm and also the corresponding part of the right arm above the elbow. In the ulnar side of the right hand just as in the ulnar side of the left hand, touch, pain, heat and cold were felt as well as in the area of the thumb. This greater loss of objective sensation in the right side corresponded to the greater sympathetic palsy of the right side, and with the numbness in the same parts and the previous pain in these parts was indicative of root rather than cord damage. Sense of position and passive movement was normal in each little finger and wrist. Vibration was normal on the ulnar side at the wrist and elbow. Hyperesthesia to the slightest contact, even of such extent as to produce dread of an object like a cloth being held near the trunk, had entirely disappeared from the upper thoracic region above the area corresponding to the level of the lesion. Within the last two days he had complained of a slight discomfort from contact on the right side in the upper thoracic region, worse at night, when his mind was not diverted, and at no time of much importance. Sensation for touch, pain, heat and cold was impaired but not lost from about the second rib to about the fifth intercostal space on each side, and they seemed to be a little more impaired on the left side of the trunk; but this was not entirely certain. It is to be remembered that the sympathetic fibers for the eye probably leave the cord through the anterior first thoracic root. The objective disturbance of sensation corresponded more to the second thoracic distribution.

Summary: Following section of the anterolateral columns on both sides at the level of the first thoracic segment, the distressing hyperesthesia complained of before the operation disappeared. There were transitory pains in the upper extremities but these gradually disappeared. An area of hypesthesia persisted along the ulnar aspect of the forearm and hand of each side. Following the operation sympathetic ocular phenomena were observed.

DISCUSSION

DR. BERNARD SACHS, New York: May I ask for a little additional information regarding the kind of cases in which operation was performed? Were they cases of tumor somewhere in the spinal cord or in the spinal canal? Moreover, was this attempt made in cases of tabes, and what was the nature of the case in which the pain was so extreme that the necessity arose to operate on the patient for the diminution of the pain?

Was the operation undertaken without any attempt to remove the tumor in those cases in which there was clearly a spinal tumor?

I have seen extremely few cases of malignant or other disease of the spinal cord in which the pain was so pronounced that I would have considered a serious operation for the relief of pain.

DR. M. ALLEN STARR, New York: I should like to ask whether subsequent to this operation any trophic disturbances appeared in the areas of the skin which were deprived of pain and temperature sense. We are familiar with the fact that in cases of syringomyelia such trophic disturbances occur, which have been ascribed partly to the loss of pain and temperature sense.

DR. SMITH ELY JELLIFFE, New York: Dr. Starr's question leads me to ask another one in which physiologic problems may enter the field of discussion. Did Dr. Spiller or Dr. Frazier make any inquiries as to the possibilities of metabolic pathways traversing in the anterior columns? Were finer tests of the kidney or liver functions investigated? Was there any evidence to show that some of the pathways that are utilized in the integration of visceral function, such as the experimental work of Brugsch, Dresel and Lewy² have shown, might pass up in that region.

DR. CHARLES H. FRAZIER, Philadelphia: The value of the communication by Dr. Spiller and myself seems to lie in the physiologic results or observations which have been drawn from the cases in which we have operated.

Two or three years ago I reported to this Association the results of the first series of chordotomies which I had performed in the neurosurgical clinic of the University Hospital, and I was quite confident at that time that the results of the operation justified the undertaking, in so far as the measure of relief was concerned. In its conception, chordotomy seems to be quite comparable to Dr. Spiller's original suggestion for avulsion of the sensory root of the gasserian ganglion in the treatment of trigeminal neuralgia, and in their effect the two operations are quite similar.

With regard to the technic: One operation is easier of performance than the other; since in avulsion of the sensory root the whole root must be sacrificed, whereas in the chordotomy the operator must exercise his judgment as to the amount of chord tissue to be sectioned. We know that in different persons

2. Dresel and Lewy: Die Zuckerregulation bei Paralysis Agitans Kranken, *Ztschr. f. d. ges. exper. med.* 26:95, 1922. Die Widal Leberfunktionsprüfung bei Paralysis Agitans Kranken, *ibid.* 26:87, 1922. Jelliffe and White: *Diseases of the Nervous System*, Ed. 4, 1922.

the size of the cord varies and even in the same person the size of the cord varies at different levels. It is for that reason that we have thought it advisable to recommend section of the cord at a given level, and for various reasons we recommend that the section be made at the level of the fifth thoracic segment.

Those who contemplate the performance of this operation should provide themselves with two special instruments. One is a hook which we have devised for the purpose of grasping and including only the anterolateral column of the cord and fixing the tissue so that the section may be made without undue trauma; the other is a special knife, so constructed that the section may be made without any technical difficulty, the blade being at an obtuse angle to the shaft.

DR. ERNEST SACHS, St. Louis: I have had opportunity to perform this operation on three or four patients, two of which were tabetics. The first patient was not relieved, this probably having been due to the fact that the section was not performed high enough.

The most satisfactory case I have had was one of carcinoma of the prostate, with metastasis in the hip joint, a case which seemed hopeless. The man was in excellent physical condition and would have been able to walk around had it not been for his intractable pain. Operation in this case relieved the patient completely, so that he was able to resume his work. He is still carrying it out six or seven months after the operation, but it is difficult to state how long he will continue to be relieved.

In the last case I made use of Dr. Frazier's hooks, and they unquestionably make the operative procedure very much simpler. I think the operation has a great field of usefulness in cases of carcinoma with metastasis to the spine, but I should hesitate to operate in tabetic patients.

DR. SPILLER, in closing: In reply to Dr. Sachs: We mentioned that in four of these cases there were malignant growths, definitely shown to be malignant either by operation or by the roentgen-ray findings. In two there were injuries received in France, with intractable and intense pain as a result, and in one, a woman, there was intense pain of long duration possibly from disease in or about the bladder. These were suitable cases for chordotomy. Of course, it would be an entirely unjustifiable procedure to perform a chordotomy if there were a possibility of removing a tumor; and we have never attempted it if there was the slightest chance of removing a tumor from the cord.

We have had no cases of tabes in this series.

Dr. Sachs says he has not seen many cases of severe pain in malignant tumor. We have observed most intense pain in malignant tumor of the vertebral column and great relief after chordotomy.

In reply to Dr. Starr's question whether there were trophic disturbances after the operation: We have not observed any changes in the skin or in the growth and appearance of the nails, but in two cases bedsores resulted. In one of these there was unquestionably implication of the crossed pyramidal tracts, while in the other the symptoms did not indicate such implication.

In regard to Dr. Jelliffe's question whether metabolic pathways were studied: We have not made a careful study of these, nor have we observed anything resulting from this operation that might point toward metabolic changes. One might expect hematuria possibly, but there was nothing of that kind in any of these cases; there was nothing that called our attention to any disturbances of the viscera.

CLINICAL NOTES ON THE PATHOLOGY IN A CASE
OF EPIDEMIC ENCEPHALITIS COMPLICATED
BY A PSYCHOSIS *

GEORGE B. HASSIN, M.D.

Associate Professor of Neurology, College of Medicine, University of Illinois;
Histologist to State Psychopathic Institute, Attending
Neurologist, Cook County Hospital

CHICAGO

AND

D. B. ROTMAN, M.D.

Senior Physician, Chicago State Hospital

DUNNING, ILL.

While instances of epidemic (lethargic) encephalitis complicated by various psychoses are by no means rare, studies of the pathology of such cases, except those of so-called striatal and pallidal syndromes, are exceedingly scarce.

REPORT OF A CASE

History.—A colored woman, 28 years of age, admitted to the Cook County Hospital on Jan. 7, 1921, complained of pains in the arms and shoulders, tremor of the left upper extremity and occipital headache. The pain was "stinging," shooting from the shoulders, especially the left, down to the fingers, while the tremor was of the "intention" variety. Both the pain and the tremor were of six days' duration. The headache dated back to an attack of influenza about eight months before.

Physical Examination.—Examination revealed jerkings and tremor of the left upper extremity, at times also of the right, and marked hyperesthesia of the neck, arms and chest. The heart, lungs and genito-urinary organs were normal. The temperature was 99.8, the pulse 82, respiration 24. The spinal fluid showed 20 cells (small lymphocytes), no globulin and a negative Wassermann reaction.

Course of Illness.—About a week later there was internal strabismus; the patient complained of diplopia vertigo and "ringing in the head." Twitchings in the left side of the face, drowsiness, hallucinations of vision and a masklike face appeared. The pupils were equal with sluggish reaction to light and absence of reaction in accommodation; there was slight ptosis of the left lid, but the strabismus disappeared. The temperature, which had ranged between 100.4 and 102 F., became normal, and the patient, feeling quite comfortable, left the hospital on March 8, two months after admission.

Readmitted about two weeks later, she was noisy, violent, restless and resistive. She was transferred on April 1, 1921, to the Psychopathic Hospital and thence the next day to the Chicago State Hospital, where she died on Jan. 22, 1922.

During the two weeks at home she had developed a somewhat indefinite psychosis. She was for the most part in fear of devils and snakes; believed that she was to be poisoned; was prayerful; thought she was dead and in another world. She was confused and attempted to jump through a window;

*From the Pathology Laboratory of the Illinois State Psychopathic Institute.

she also made a violent attack on her husband and children. But she had a short period of paretic-like expansiveness in which she believed that she and the entire family were going to visit relatives in the South, and that she was going to do everything in grand style; she wanted all of them to have new outfits of clothing, etc. The money was to come from a drawer which was filled with thousands of dollars, placed there by God. God was telling her at that time that she could have whatever she wished.

On admission the neurologic picture had faded, a somewhat sluggish pupillary reaction and some bilateral divergent strabismus remaining. She presented a picture of extreme episodic excitement and proved to be a most violent patient. Although she was a very small woman, it was frequently necessary to use physical restraint. She would often attack any one within reach and appeared to be unclear at the time of these attacks. The only thing that her excitement can be likened to is the extreme furor of an epileptic. She would dash through a window, cutting herself severely, just as an epileptic does.

At other times her reactions were of quite a different kind. She would cry, ask forgiveness for her conduct, and beg those about her to be good to her. At these times her contact with environment was apparently good. She soon learned the names of all who came near her and would even pick up their first names. She was in an unquestionably delirious state at times, when she would confuse those about her with members of her own family. At no time was her stream of speech a clear one, nor could it ever be actually directed.

She remained in practically the same condition for eight months, during which time her general physical condition remained stationary. Then her excitements became more intense and more frequent. Finally, during these excitements the patient began, without explanation, to mutilate herself. One of the results of this self-mutilation was the production of a rectovaginal fistula. Attempts to treat this fistula proved ineffective because of the interference of the patient. On Jan. 17, 1922, her bed was found covered with blood. She had succeeded in pulling a loop of intestine through the fistula, and there was a complete tear through the intestine. She did not complain and made no explanations, exhibited no signs of shock and did not seem distressed. She was immediately operated on and died two days later with peritonitis.

Histopathologic Report.—Microscopic examination of the apparently normal brain revealed the following:

Ganglion Cells and Nerve Fibers: Many ganglion cells, especially in the cortex, appeared quite normal. Some showed marked destruction; some neuronophagia, chromatolysis or cell sclerosis, especially in the basal ganglions and midbrain. Throughout the cortical layers which retained their general architecture, as well as in the granular layer of the cerebellum, in the basal ganglions and midbrain the nerve cells exhibited an enormous accumulation of lipoids. Lipoids were also found in the ependymal cells of the aqueduct of Sylvius and of the lateral ventricles. Especially abundant were lipoids in the optic thalamus, substantia nigra, pons and corpus striatum, while the globus pallidus, nucleus ruber and the occipital lobe showed comparatively little fat in the cells, but large amounts in the blood vessels. The nerve fibers, the axons and myelin showed no changes whatever.

Glia: Proliferation of glia nuclei was quite marked in the deeper strata of the cortex, also in the optic thalamus, putamen and the region of the aqueduct. In the latter areas, as well as in the nucleus dentatus cerebelli, there were numerous cytoplasmic glia cells, some containing two nuclei, and many rod

cells (Stäbchenzellen) considered by some gliogenous, by some mesodermogenic elements. Stained with scarlet red the glia cells revealed minute droplets of lipoids.

Blood Vessels: In the midbrain and the basal ganglions (especially in the optic thalamus) some vessels were mildly infiltrated with hematogenous elements (lymphocytes, plasma cells). In other places the dilated Virchow-Robin spaces contained vacuolated bodies filled with yellow granules (in specimens stained with toluidin blue or methyl blue eosin). Such vacuolated bodies much resembled gutter cells packed with lipoids, and were also found in the cortex (especially in the occipital region). Much more marked were so-called proliferative or productive phenomena. Mild and unusual in the cortex, they were much in evidence in the subcortical regions, and especially in the nucleus caudatus, pons and around the aqueduct, where they showed as hypertrophied deeply stained endothelial cells, buddings and new formed capillaries. Much more common and widespread were infiltrations of the small and larger vessels with lipoids which in some instances (basal ganglions and midbrain) were present in large amounts.

Pia-Arachnoid and Choroid Plexus: The pia-arachnoid was markedly infiltrated with mesothelial cells, fibroblasts, some lymphocytes and modified plasma cells. Over the occipital and parietal lobes the distended meshes of the pia-arachnoid contained typical ganglion cells, some surrounded by lymphocytes, some perfectly intact, with a distinct nucleus and processes. Such a peculiar phenomenon observed by one of us (G. B. H.) in many other conditions is recorded here as a curiosity. This heterotopia evidently was an artefact. The arachnoid villi (pacchionian bodies) were greatly hypertrophied, hyperplastic. The tuft cells of the choroid plexus contained an abundance of large granules and vacuoles, a well developed nucleus rich in chromatin, while the perivascular stroma was densely infiltrated with cell bodies, some of which much resembled mesothelial or endothelial cells. Both the pia-arachnoid and the choroid plexus contained large amounts of lipoids.

Summary of the Microscopic Findings.—1. Mild inflammatory infiltrative phenomena in the basal ganglions and midbrain.

2. Widespread proliferative or productive vascular changes throughout the brain—pronounced in the basal ganglions and the midbrain, but rare and mild in the cortex.

3. Diffuse parenchymatous changes with a vast accumulation of lipoids in the nerve cells, blood vessels, choroid plexus, pia-arachnoid and to a lesser extent in the glia.

4. Proliferation of glia in the basal ganglions.

COMMENT

We have in this case a combination of diffuse inflammatory and degenerative phenomena. Both were especially in evidence in the basal ganglions and midbrain. But the infiltrations were not so pronounced as in acute cases of epidemic encephalitis, while the proliferative changes were not so marked as in so-called productive encephalitis (caused, for instance, by lead or arsenic). Resembling in the combinations of both inflammatory and degenerative changes the findings in general paralysis of the insane, this case differs from the latter in the comparatively mild involvement of the cortex. The inflammatory

changes in the basal ganglions and midbrain could perhaps explain the nervous manifestations in our patient (ocular paralysis, parkinsonian face), while the diffuse degenerative phenomena in the cortex were most likely responsible for the patient's mental condition. It is obvious that degenerative lesions are a serious complication of epidemic encephalitis, much more serious than the inflammatory changes. For the latter may subside or disappear entirely, leaving no permanent disability, in contrast to a degenerative process, which, however mild, may produce more or less troublesome complications of which the most dangerous are mental disturbances.

As the cause of the degenerative changes is most likely toxins elaborated by the virus of epidemic encephalitis, and as they may prove very virulent and dominate the pathologic condition, it follows that the prognosis of epidemic encephalitis is always doubtful.

In conclusion we wish to call attention to a purely histopathologic feature: fat accumulation in the ependymal cells of the lateral ventricles, in the pia-arachnoid and tuft cells of the choroid plexus. This has been discussed by one of us (G. B. H.) in previous contributions and is pointed out only to emphasize the fact that such fatty infiltrations occur in any degenerative condition.

NEUROSYPHILIS AMONG THE CHINESE

WITH FINDINGS IN SIXTY-FIVE CASES

WILLIAM G. LENNOX, M.D.

PEKING, CHINA

INCIDENCE OF SYPHILIS

Practitioners in China agree that syphilis is prevalent among the Chinese. In order to secure statistical information on the subject, a request for annual reports was made of all hospitals in China. Of the reports secured, twenty-nine from fifteen different hospitals¹ contained lists of diagnoses made on inpatients. Three of these hospitals in addition published diagnoses made on outpatients.

For comparison, data from the annual reports of nine hospitals² in America was compiled. Statistics from these two groups of hospitals are presented in Tables 1, 2 and 3. Lack of uniformity in diagnostic

TABLE 1.—INCIDENCE OF SYPHILIS AMONG HOSPITAL PATIENTS
IN CHINA AND AMERICA

General Hospitals*	Inpatients			Outpatients		
	Total Number of Patients	Number of Cases of Syphilis	Percentage of Cases of Syphilis	Total Number of Patients	Number of Cases of Syphilis	Percentage of Cases of Syphilis
China.....	35,525	2,906	8.4	57,242	3,513	6.1
America.....	100,143	2,516	2.5	68,402	2,063	3.1

* In these tables data concerning inpatients are from fifteen hospitals in China and nine in America, and concerning outpatients from three hospitals in each country. Patients in all departments are included.

facilities, in disease classifications used and in methods of compiling medical statistics in the various hospitals, does not permit a close comparison of figures.

It is evident from Table 1 that the incidence of syphilis among the patients of the hospitals selected is two or three times greater in China than in America.

1. The hospitals, with the number of inpatients reported (often for several successive years) are: Peking Union Medical College, 7,138; Canton Christian College, 6,976; Rankine Memorial, Ichang, 5,297; University of Nanking, 2,755; St. Luke's, Shanghai, 2,047; Church General, Wuchang, 2,017; Red Cross, Shanghai, 1,711; Wuhu, 1,393; Soochow, 1,384; Kaifeng-Hunan, 1,348; Chefoo-Shantung, 1,095; Shantung Christian College, 1,003; Tsing Wang Pu, 789; Siangtan, 560; Pei Yang, Tientsin, 318.

2. The hospitals, with the number of inpatients reported are: Johns Hopkins (whites), 6,151; Bellevue, 50,403; Presbyterian, New York, 7,324; New York, 5,944; Lakeside, Cleveland, 5,929; Peter Bent Brigham, Boston, 3,533; Pennsylvania, 5,662; University of Pennsylvania, 6,863; Massachusetts General, Boston, 8,834. The last three gave diagnoses also of outpatients.

The results of routine Wassermann tests performed on Chinese inpatients have been reported as follows: In Peking, of 494 male medical patients, 24 per cent. gave positive tests (Sia³). In Soochow, of 752 medical and surgical patients, three fourths of whom were male, the corresponding proportion was 39 per cent (Snell⁴). Similarly compiled statistics have been reported from many hospitals in America. The results of these examinations, summarized by Vedder,⁵ show from 10 to 30 per cent. of patients giving positive Wassermann reactions.

Wassermann tests performed in Peking on 800 Chinese domestic servants preliminary to employment yielded 11 per cent. positive tests (Korns⁶).

INCIDENCE OF NEUROSYPHILIS

In spite of the prevalence of syphilis among the Chinese, cases of neurosyphilis are apparently comparatively rare. The following testimony on the subject is given by men who have practiced among the Chinese for many years. Hodge⁷ saw only one or two cases of tabes dorsalis and one case of general paresis. Maxwell⁸ never saw a typical case of general paresis and only three of tabes. Reed⁹ saw only one or two cases of tabes.

Similar evidence concerning another branch of the Mongolian race is given by Montel,¹⁰ who found only one case of tabes among 100,000 patients treated in Indo-China. Personal application for information similar to that secured from hospitals in China and America was made of twenty of the leading general hospitals in Japan. It was found, however, that these hospitals do not keep a tabulated record of the diagnoses made. Thom¹¹ says that in Japan neurosyphilis is rare, but he does not cite the evidence on which the statement is based.

The comparative incidence of neurosyphilis in the two groups of hospital patients in China and America is shown in Table 2. Central nervous system involvement among syphilitic inpatients is seven or eight times more frequent in America than in China. The difference among

3. Sia, R. H. P.: Routine Wassermann Test on 502 Inpatients, China M. J. **35**:39 (Jan.) 1921.

4. Snell, J. A., and Chang, P. Y.: Report of Routine Wassermann Test at Soochow Hospital for One Year, China M. J. **35**:36 (Jan.) 1921.

5. Vedder, E. B.: Syphilis and Public Health, Philadelphia, Lea and Febiger, 1918, p. 97.

6. Korns, J. H.: Further Statistics on Communicable Diseases Among Domestic Servants, China M. J. **35**:382 (July) 1921.

7. Hodge, S. R.: Syphilis in China, China M. J. **21** (Sept.) 1907.

8. Maxwell, James: Syphilis Among Chinese, China M. J. **27** (Nov.) 1913.

9. Reed, A. C.: Nervous Diseases in China, China M. J. **29** (Nov.) 1915.

10. Montel, L. R.: Un cas de tabes chez un Annamite, Bull. Soc. méd. Chir. Indo-China, October, 1916.

11. Thom, B. P.: Strain in Spirochetes, Am. Jour. Syph. **5**:1 (Jan.) 1921.

outpatients is not so great because most of the outpatients in China are reported by one hospital in Peking, and this hospital, for reasons to be explained, treats a relatively high proportion of cases of neurosyphilis.

Additional evidence concerning the incidence of neurosyphilis is furnished by institutions for the insane. The Robert G. Kerr hospital at Canton is the only modern hospital for the insane in China. During the last seven years, 312, or 14.7 per cent., of the patients treated were diagnosed as having general paresis. Samuels¹² found only 3.4 per cent. among the Chinese in one asylum in Malay. The annual reports of thirteen hospitals¹³ for the insane in America, with admissions totaling 5,955, show an incidence for general paresis and taboparesis of 6.7 per cent. London Insane Asylums¹⁴ show a ratio for general paretics of 8 per cent., the New York State hospital a ratio of 18 per cent. for men and 13 per cent. for women (Kindred¹⁵).

TABLE 2.—INCIDENCE OF NEUROSYPHILIS AMONG HOSPITAL PATIENTS IN CHINA AND AMERICA

General Hospitals	Inpatients			Outpatients		
	Number of Cases of Neuro-Syphilis	Percentage of Patients	Percentage of Cases of Syphilis	Number of Cases of Neuro-Syphilis	Percentage of Patients	Percentage of Cases of Syphilis
China.....	150	0.4	5.1	90	0.16	2.7
America.....	1,008	1	39.8	150	0.23	7.6

Hofman,¹⁶ Woods¹⁷ and Harvey¹⁸ have presented the general physical and mental findings in some of the Robert G. Kerr hospital cases. Unfortunately, examination of the patients at this hospital has not included the Wassermann test of the blood serum or examination of the spinal fluid.

The bulk of the evidence at hand points to the comparative rarity of syphilis of the nervous system in the yellow race. Similar reports

12. Samuels, W. F.: General Paresis in the Tropics, *Indian Med. Gaz.* **51**:453 (Dec.) 1916.

13. These hospitals include eight state and one private hospital in Massachusetts, and one state institution in Connecticut, Maryland, Indiana and New Hampshire.

14. Report of the Royal Commission on Venereal Diseases, 1916, p. 137.

15. Kindred, J. J.: Venereal Diseases, Their Relation to Insanity and Nervous Diseases, *New York M. Rec.* **93**:184, 1918.

16. Hofman, J. A.: *China M. J.* **27** (Nov.) 1913.

17. Woods, A. H.: Diseases of the Spinal Cord Among the Chinese, *China M. J.* **32** (March) 1918.

18. Harvey, J. L.: Cases of General Paresis in China, *China M. J.* **34** (March) 1920.

concerning the black race have been made by several observers. At least five explanations of this apparent racial discrimination have been advanced:

1. Among the colored races there is a specific strain of spirochete which spares the central nervous system. Hofman¹⁶ says that many of his cases of general paresis in Canton occurred in steamship employees, who derived their infections from a non-Chinese source. Most of the hospitals in China reporting cases of neurosyphilis are located in coast cities which have long been exposed to western syphilization. More statistics are needed from inland China before the value of this argument as regards Chinese can be judged.

2. The colored races have a nervous system less sensitive and less susceptible to syphilitic infection than has the white race.

3. Among the colored races syphilis is a lately acquired infection and has not yet reached the attenuated form in which central nervous system damage is common. This argument would not hold for the Chinese because syphilis has been with them in all probability for many hundreds of years.

4. The colored races have not been subjected to modern antisymphilitic treatment, which, according to Fraser¹⁹ and others, leaves the spirochete, bandit-like, in the inaccessible fastnesses of the central nervous system. A possible illustrative case in our series is that of a Chinese cook for an American family, who on initial examination presented a chancre scar and a secondary eruption. He was given nineteen intramuscular injections of mercury and three intravenous injections of arsaminol, a Japanese preparation of arsphenamin. Six months after beginning treatment he became completely deaf. His ear drums were normal. Wassermann tests of the blood and spinal fluid were negative, but the spinal fluid gave a positive globulin test and contained 270 cells per cubic millimeter. The patient accepted discharge from his position rather than undergo any further treatment at the hands of the foreign physician.

5. The reputed low incidence of neurosyphilis among colored races may be more apparent than real. In support of this explanation are the following considerations: The hospitals and clinics reporting white patients with neurosyphilis are especially organized and equipped for discovering cases of this disease. A considerable proportion of these cases, one-half in some instances (Dennie and Smith,²⁰ Wile and

19. Fraser, A. R., and Duncan, A. G. B.: A Possible Explanation of the Increased Incidence and Early Onset of Neurosyphilis, *Brit. Jour. Dermat. & Syph.* **33**:251 (July) 1921.

20. Dennie, C. C., and Smith, D. O.: The Blood and Cerebrospinal Fluid in Three Hundred Known Cases of Syphilis, *Am. J. Syphilis* **2**:101 (Jan.) 1918.

Marshall²¹), which show abnormality of the spinal fluid, present no clinical signs of central nervous system involvement. Reports from China, on the other hand, have come from hospitals not so organized or equipped. Diagnoses have been made chiefly by men busy with surgery, on the basis of well marked physical signs alone. Pupillary abnormalities are more easily missed in dark-eyed races, and often the Chinese do not present the symptoms of nervous disorder which are typical for white patients. For example, I have found that the complaint of "lightning pains" among Chinese tabetics is rare.

The situation is illustrated in the case of the Peking Union Medical College hospital. The incidence of neurosyphilis among syphilitic patients in this hospital is about six times that reported by the fourteen other hospitals in China. Before the institution of a careful search for these cases, they had been seen no more frequently in Peking than elsewhere, but during a three year period in which a special syphilis clinic was maintained and free use made of the Wassermann laboratory and lumbar puncture needle, the number of cases among both inpatients and outpatients steadily increased. Thus, nineteen new patients with neurosyphilis were admitted to the medical ward during the first fourteen months and an equal number during the last four months of the period. During the whole three years, neurosyphilitic cases formed about 3.3 per cent. of the new medical admissions in Peking, against about 1.6 per cent. in the previously named hospitals in America.

With respect to outpatients of all departments, the percentage of cases of neurosyphilis for the three successive years was 0.11 per cent., 0.22 per cent. and 0.4 per cent., respectively. The average for the 35,000 patients seen during the whole period, 0.23 per cent., is the same as the average proportion reported for 68,000 patients from the three hospitals in America.

This rapid increase of cases was due in part to former patients and to physicians in other hospitals who referred cases for treatment. In larger part it was due to the well recognized relation between seeking and finding. Fewer cases in the clinic went unrecognized.

This fifth explanation probably will not account for all the observed racial differences in the incidence of neurosyphilis. Thus, Zimmermann²² found that neurosyphilis among patients in the same clinic was twice as common among whites as among blacks. It seems probable, however, that when hospitals in China have staffs and equip-

21. Wile, V. W., and Marshall, C. H.: A Study of the Spinal Fluid in One Thousand Six Hundred and Eighty-Nine Cases of Syphilis in All Stages, *Arch. Dermat. & Syph.* 3:272 (March) 1921.

22. Zimmermann, E. L.: A Comparative Study of Syphilis in Whites and in Negroes, *Arch. Dermat. & Syph.* 4:75 (July) 1921.

ment which will make thorough search possible, the present apparent differences in the nervous manifestations of syphilis in the white and yellow races will be markedly decreased.

DISTRIBUTION OF VARIOUS LESIONS

The striking difference in the distribution of lesions is the high proportion of cord cases among the Chinese (Table 3).

Of the 26 per cent. with central nervous system lesions of vascular origin, six out of seven present a paraplegia due to transverse myelitis of the cord. Cases of general paresis, in contrast, are rare, possibly due in part to the fact that they present less urgent symptoms and less obvious physical signs.

ANALYSIS OF CASES

Since no series of cases of neurosyphilis in Chinese confirmed by spinal fluid examinations has been found in the literature,²³ it seems

TABLE 3.—INCIDENCE OF VARIOUS FORMS OF NEUROSYPHILIS AMONG HOSPITAL INPATIENTS IN CHINA AND AMERICA

General Hospitals	Number of Cases				Percentage of Cases			
	General Paresis	Tabes Dorsalis	Cerebro-spinal	Lesions of Vascular Origin	General Paresis	Tabes Dorsalis	Cerebro-spinal	Lesions of Vascular Origin
China.....	5	51	55	39	3	34	37	26
America....	481	299	223	—	48	39	22	—

worth while to record the principal findings in the group of sixty-five cases seen during the three year period in Peking. Some of these cases were under the charge of colleagues, Dr. Korn and Dr. Smyly. All the patients had definite physical signs of the condition, and in addition abnormality in the Wassermann reaction, cell count or globulin content of the spinal fluid. The series is too small to permit discussion of the interesting question of racial differences in symptoms and signs.

All the cases occurred in men as only men were admitted to the hospital. Twenty-six per cent. of these patients and 13 per cent. of all inpatients were soldiers. Soldiers were found particularly likely to have transverse myelitis, a fact which has been noted in other countries. All but six of the sixty-five gave a history of a penal sore, contracted on the average 5.4 years previously (10 years for patients with tabes and 3.3 years for patients with myelitis). For a few of the cases, the

23. Since this paper was written the following article has appeared: Woods, A. H.: Types of Cerebro-Spinal Syphilis in China, *China M. J.* 36:206 (May) 1922.

Wassermann test was not available. Of the others, 75 per cent. gave a positive reaction in the blood serum and 65 per cent. in the spinal fluid. In 65 per cent., the spinal fluid globulin was increased; in all cases the cell count was above normal. The average count on the initial puncture was 87 cells per cubic millimeter.

The distribution of cases was: transverse myelitis 29, tabes dorsalis 16, cerebrospinal 14, hemiplegia 5, general paresis 1. The average initial cell count in the spinal fluid in these groups was: transverse myelitis 82, tabes 39, cerebrospinal 115, hemiplegia 73, general paresis 50. Besides the twenty-nine patients with transverse myelitis with positive evidence of syphilis, there were in the hospital during this period ten other patients with paraplegia not showing such evidence. Four fifths of the patients with myelitis due to syphilis had urinary retention or incontinence. Two thirds of the patients with cerebrospinal syphilis showed optic neuritis or atrophy. In one case there was a binasal hemianopsia. The frequency of these complications is perhaps due to the urgency of the symptoms.

Whatever is done for Chinese patients must be done quickly, or they go elsewhere. For this reason, and because of the comparatively small amount of the physician's time and patient's money required, intraspinal injections of mercurialized serum were given. Thirty-six of the patients received from one to nine such injections.

Too few of the patients remained under observation long enough to allow judgment concerning the value of this form of treatment. All cases showed prompt fall in spinal fluid cell count, but in only two did positive spinal fluid Wassermann tests become negative. Whether as a result of treatment or not, one or two of the apparently hopelessly paralyzed patients improved.

A soldier of 35, who had had a primary sore seven months previously, suddenly developed numbness of the legs and retention of urine. After eight days, he entered the hospital. At that time he had exaggerated knee reflexes with ankle clonus and a positive Babinski sign. Blood serum and spinal fluid both yielded four plus Wassermann reactions. The spinal fluid showed increased globulin and 900 cells per cubic millimeter. He was able to walk on admission, but complete paralysis of the legs came on rapidly, together with complete retention of urine. During 125 days in the hospital, he received seven intravenous injections of arsaminol and nine intraspinal injections of mercurialized serum. Successive spinal fluid counts were 900, 495, 90, 63, 30, 50, 20, 10, 5 and 3. Fluid Wassermann tests became negative, that of the blood serum remained two plus. Though increased knee reflexes and ankle clonus persisted, when he left the hospital he was able to walk and to urinate.

SUMMARY

Testimony of individual observers and reports of general hospitals agree that in China syphilis is relatively more common and neurosyphilis relatively less common than in America.

One explanation for this apparent racial discrimination is the probability that, for reasons named, many cases of neurosyphilis among the Chinese have been overlooked. In a teaching hospital in Peking, search for these cases revealed a proportion comparable with that found in teaching hospitals in America.

An unusual proportion of the Chinese patients have cord lesions of vascular origin.

Sixty-five cases of neurosyphilis are reported in which spinal fluid examination confirmed the clinical diagnosis.

A STUDY OF FOUR CASES OF GLOSSOPHARYNGEAL NEURALGIA *

JOHN B. DOYLE, M.D.

ROCHESTER, MINN.

The anatomy of the glossopharyngeal nerve is a matter of dispute, and its exact relationship to the tenth and eleventh cranial nerves is matter for conjecture. All authorities differ with regard to the exact details of its central and peripheral connections.

According to Ranson,¹ the glossopharyngeal is a mixed nerve made up of general and special visceral afferent fibers and general and special visceral efferent fibers. The general and special afferent fibers have their cells of origin in the petrous ganglion. Their central branches terminate in the solitary tract and its nucleus. The general visceral efferent fibers arise from the cells of the inferior salivatory nucleus of the medulla oblongata. The special visceral afferent fibers arise in the nucleus ambiguus, the medullary continuation of the dorsolateral cell group of the anterior column of the cord.

Superficially the glossopharyngeal nerve takes its origin from the rostral end of the posterolateral sulcus of the medulla oblongata in line with the tenth and eleventh nerves. Like the vagus, the glossopharyngeal nerve has two ganglions. The dorsal root ganglion, the superior ganglion, is at the upper orifice of the jugular foramen; the trunk ganglion, the petrous ganglion, is in a small depression at the lower orifice of the jugular foramen. From the latter, branches of communication pass to the auricular branch of the vagus, to the jugular ganglion and to the sympathetic.

As the facial nerve emerges from the stylomastoid foramen it receives a branch of communication from the trunk of the ninth nerve, which passes through the posterior belly of the digastric. The first branch of the ninth nerve, according to Gray,² is the tympanic nerve, the nerve of Jacobsen, which forms the tympanic plexus from which arise the lesser superficial petrosal, a branch to the greater superficial petrosal, and branches to the tympanic cavity. The lesser superficial petrosal passes to the otic ganglion, a small, oval flattened mass below the foramen ovale on the medial aspect of the mandibular nerve, which

* From the Section on Neurology, Mayo Clinic.

* Read before the Minnesota Neurological Society, Rochester, Minn., May 13, 1922.

1. Ranson, S. W.: *The Anatomy of the Nervous System from the Standpoint of Development and Function*, Philadelphia, W. B. Saunders Company, 1921.

2. Gray, H.: *Anatomy of the Human Body*, Ed. 20, Philadelphia, Lea and Febiger, 1918, pp. 906-909 and 891-893.

surrounds the nerve to the internal pterygoid muscle. This ganglion receives fibers from the nerve to the internal pterygoid, from the seventh nerve, and from the sympathetic fibers accompanying the middle meningeal artery. The preganglionic fibers are derived from that part of the glossopharyngeal nerve which originates in the inferior salivatory nucleus. The postganglionic fibers pass through the auriculotemporal nerve to the parotid gland.

The carotid branches of the ninth nerve communicate with the sympathetic on the carotid artery. The pharyngeal branches are composed of three or four filaments which unite posterior to the middle constrictor of the pharynx with the vagus and sympathetic to form the pharyngeal plexus. The muscular branch supplies the stylopharyngeus muscle. The tonsillar branches supply the palatine tonsil, the soft palate, and the fauces, and communicate with the palatine nerves. The lingual branches terminate in the papillae vallatae and the mucous membrane of the base of the tongue.

In 1918, Vernet,³ in discussing the syndrome of the jugular foramen, said that a destructive lesion of the ninth nerve causes paralysis of the superior constrictor of the pharynx which results in difficulty in swallowing solid food. Paralysis of the tenth nerve results in anesthesia of the soft palate and the posterior wall of the pharynx. Destruction of the accessory part of the eleventh nerve leads to paralysis of the soft palate and larynx, while destruction of the spinal portion produces paralysis of the sternocleidomastoid and trapezius muscles.

Trifacial neuralgia has long been accepted as a clinical entity. The characteristic paroxysmal pain, familiar to all neurologists and to most physicians, occurs in the area of distribution of one or more of the three divisions of the trigeminal nerve.

In 1907 and 1908, Hunt⁴ discussed in detail inflammations of a herpetic nature, involving the ear and adjacent regions. He attributed them to an involvement of the geniculate ganglion similar to involvement of the gasserian ganglion and dorsal root ganglions in herpes zoster. The pain was different from that typical of tic douloureux.

In 1908, Orbison⁵ reported a case of herpes of the tympanic membrane and endeavored to prove the condition "due to zosteroid affection of the petrosal ganglion."

3. Vernet, M.: Syndrome du trou déchiré postérieur (paralysie des nerfs) glosso paryngien, pneumogastrique (spinal), *Rev. neurol.* **34**:117-148, 1918.

4. Hunt, J. R.: On Herpetic Inflammations of the Geniculate Ganglion. A New Syndrome and Its Complications, *J. Nerv. & Ment. Dis.* **34**:73-94, 1907. A further Contribution to Herpetic Inflammations of the Geniculate Ganglion, *Am. J. M. Sc.* **136**:226, 1908.

5. Orbison, T. J.: Herpes of the Membrane Tympani: Due to Zosteroid Affection of the Petrosal Ganglion, *J. Nerv. & Ment. Dis.* **35**:500-506, 1908.

In 1910, Mills⁶ refuted Hunt's arguments concerning the nature of the afferent fibers of the facial nerve and attempted to show that the function of the nerve was chiefly gustatory and that only a vestigial remnant of common sensation remained over a small portion of the anterior part of the tongue and a narrow strip in the auricle.

Lesions of the glossopharyngeal nerve, experimental or acquired, are extremely rare. In 1896, Meyer⁷ described the changes in the taste buds of foliate type, of the tongue of the guinea-pig, subsequent to section of the ninth nerve.

In 1919, Neve⁸ described a case of what he termed herpes zoster of the glossopharyngeal nerve. The patient had peripheral facial nerve palsy on the left, impairment of the eighth nerve, vesicular eruption over the left half of the soft palate, and pain posterior and inferior to the left ear and down the left side of the neck.

Oppenheim⁹ described a case of paralysis of the ninth nerve in a woman. Objectively she had thermesthesia, paresis of the soft palate and pharyngeal muscles, and loss of taste in the soft palate, pharynx and posterior part of the tongue. Tactile sense was preserved intact.

In 1910, Weisenburg¹⁰ described a case of cerebellopontile tumor diagnosed for six years as tic douloureux. Frazier cut the sensory root of the fifth nerve and excised most of the gasserian ganglion relatively early, to relieve what appeared to be trifacial neuralgia. Following this operation the patient complained of pain in the right side of the throat radiating into the ear and neck and occasionally to the shoulder, and later he had severe pain at the root of the tongue and in the right side of the pharynx. This pain was paroxysmal and was set up by the contact of food or other foreign material with the mucous membrane of the pharynx. He also complained of pain in the lower right side of the face. It should be emphasized that the character of the pain differed after the ganglion operation in that before the pain was in the whole face as well as in the throat. Repeated pharyngeal and laryngeal examinations revealed excessive tenderness on the right side, but no weakness of any of the muscles of that region. At necropsy a tumor was discovered in the right cerebellopontile angle lying directly on the

6. Mills, C. K.: The Sensory Functions Attributed to the Seventh Nerve, *J. Nerv. & Ment. Dis.* **37**:273-284 and 354-377, 1910.

7. Meyer, S.: Durchschneidungs-versuche am Nervus Glosso-pharyngeus, *Arch. f. mikrosk. Anat.* **48**:143-145, 1896.

8. Neve, G. T.: Herpes Zoster of the Glossopharyngeal Nerve, *Brit. M. J.* **2**:630, 1919.

9. Oppenheim, H., quoted by Weisenburg; T. H.: *J. A. M. A.* **54**:1600 (May 14) 1910.

10. Weisenburg, T. H.: Cerebello-Pontile Tumor Diagnosed for Six Years as Tic Douloureux: The Symptoms of Irritation of the Ninth and Twelfth Cranial Nerves, *J. A. M. A.* **54**:1600-1604 (May 14) 1910.

sensory and motor roots of the fifth nerve. The ninth and tenth nerves, which were very prominent, stretched over the lower portion of the tumor. On account of the close relationship of the peripheral fibers of the fifth and ninth nerves, Weisenburg interpreted the pain which persisted in the area of distribution of the fifth nerve after resection of the major portion of the gasserian ganglion as a result of irritation of the ninth nerve. The pharyngeal pain was also attributed to irritation of the ninth nerve.

In 1920, Sicard and Robineau¹¹ described three cases of what they termed "algie vélo-pharyngée essentielle." The first two cases were seen in the French army in 1916 and 1917, respectively. In one case the pain was right sided; in the other, left. In neither case could a cause be established. There was no antecedent history of syphilis, and the condition did not respond to treatment for syphilis, to sedatives or physical agents. In both cases the pain had persisted over a period of several years; it was not continuous, but recurred frequently day and night in extremely acute paroxysms on the least movement of mastication, deglutition or speech. Occasionally the paroxysms occurred spontaneously. The patients developed suicidal tendencies on account of the apparent incurability of the condition.

The treatment advised consisted in section of the glossopharyngeal nerve and the pharyngeal branches of the vagus, with ablation of the superior cervical sympathetic ganglion. These two patients were operated on by surgeons unfamiliar with surgical procedures in the neck. In one the ninth nerve was cut, the sympathetic ganglion was removed, and the trunk of the vagus severed; in the other the ninth and twelfth nerves and the pharyngeal branches of the tenth nerve were cut, and the sympathetic ganglion was removed. Both of the patients convalesced rapidly. The first patient developed unilateral palsy of the vocal cord; the second, hemiatrophy of the tongue.

Sicard's third case was that of a woman, aged 50 years, in whom he successfully cut the ninth nerve and the pharyngeal branches of the tenth, and ablated the superior cervical sympathetic ganglion. During operation the trunk of the vagus was pulled, resulting in paresis of the homolateral vocal cord; this condition, however, was steadily improving. As in the first two instances, all pain disappeared immediately following operation. The first patient has been completely relieved for four years and the second for three.

Harris¹² has recently described two cases of a rare form of chronic paroxysmal neuralgia, which he termed glossopharyngeal neuralgia.

11. Sicard, R. and Robineau: *Algie vélo-pharyngée essentielle*. *Traitement chirurgical*, *Rev. neurol.* **36**:256-257, 1920.

12. Harris, W.: *Persistent Pain in Lesions of the Peripheral and Central Nervous System*, *Brain* **44**:557-571, 1921.

The pain, according to him, characteristically starts in the throat in the region of the tonsil and anterior pillar and radiates to the ear and upper part of the neck. The first case was that of a man, aged 40 years, whom he had first seen ten years before, when he successfully injected the third division on the affected side without influencing the pain. The second patient was a woman, aged 87 years, who had suffered from the condition for ten years. Harris was convinced of the involvement of the ninth nerve in these cases; he had seen a man with similar pains and intense hyperesthesia of the side of the neck, following a recurrence of epithelioma of the tonsil seven years after extirpation of a malignant vocal cord.

REPORT OF FOUR CASES OF GLOSSOPHARYNGEAL NEURALGIA OBSERVED
IN THE MAYO CLINIC BETWEEN MARCH 14 AND JUNE 1, 1922

CASE 1.—History.—Mr. W. H. L., a farmer, aged 63 years, came to the Clinic, March 14, 1922, complaining of pain in the throat and right ear. In 1912, he had had herpes zoster just above the left iliac crest. Five years before, after taking a drink of cold water he had had a sharp paroxysmal pain in the region of the right ear, associated with excessive tenderness of the right auricle. Similar seizures occurred sporadically until 1918, when his tonsils were removed. He was completely relieved for about three years, when, for six months, he had mild paroxysms. Following the extraction of several infected teeth in the fall of 1921, he was relieved until the last week in February, 1922, when the pain recurred while he was drinking water. At the time he came to the Clinic he had great difficulty in obtaining sufficient nourishment, owing to the pain induced by drinking and masticating. The pain was paroxysmal, sharp and agonizing; it arose in the right faucial region and radiated laterally to the area anterior and posterior to the right ear. It lasted from thirty to ninety seconds and was definitely increased in frequency by talking and chewing. At the onset he had attacks during the night; during the two weeks preceding his examination his distress had been chiefly diurnal.

Examination.—The physical examination was negative except for slight arteriosclerosis and moderate enlargement of the prostate. The systolic blood pressure was 118, the diastolic 80. Vision was 6/6 (with correction) in both eyes. Both pupils were slightly irregular. Reflexes and fields were normal. Both fundi were negative, except for slight choroidal sclerosis. A few peripheral lenticular opacities were noted. The Wassermann reaction was negative, as were the urinalysis, blood counts and stereoscopic roentgenograms of the head. On examination of the larynx and nasopharynx a trigger area was discovered deep in the hypopharyngeal region. The only neurologic findings were a slight to moderate diminution in perception of vibration and slight incoordination of both lower extremities.

Diagnosis: The neurologic diagnosis was trifacial neuralgia involving the auriculotemporal branch of the right mandibular division.

Treatment and Course.—On March 23, 1922, the sensory root of the right gasserian ganglion was cut, and the motor root preserved. The patient was greatly relieved. On April 8, there was complete anesthesia for tactile, pain and temperature senses over the area of distribution of the right trigeminal nerve. Sensation to touch and pain was normal in the external auditory canals and over the external aspects of both tympanic membranes. On April 14, the

patient returned to the Clinic. The pain had been recurring for the last forty-eight hours. During examination the paroxysms were almost continuous. Morphin, hypodermically, gave but little relief. On April 25, the glossopharyngeal nerve was evulsed and the pharyngeal branch of the vagus cut. For the last six weeks the patient has not had a recurrence of the pain. For a few days after operation he complained of a sense of fulness in the right side of the pharynx with a slight dysphagia. He has complete anesthesia to pain, temperature and touch over the area of cutaneous distribution of the

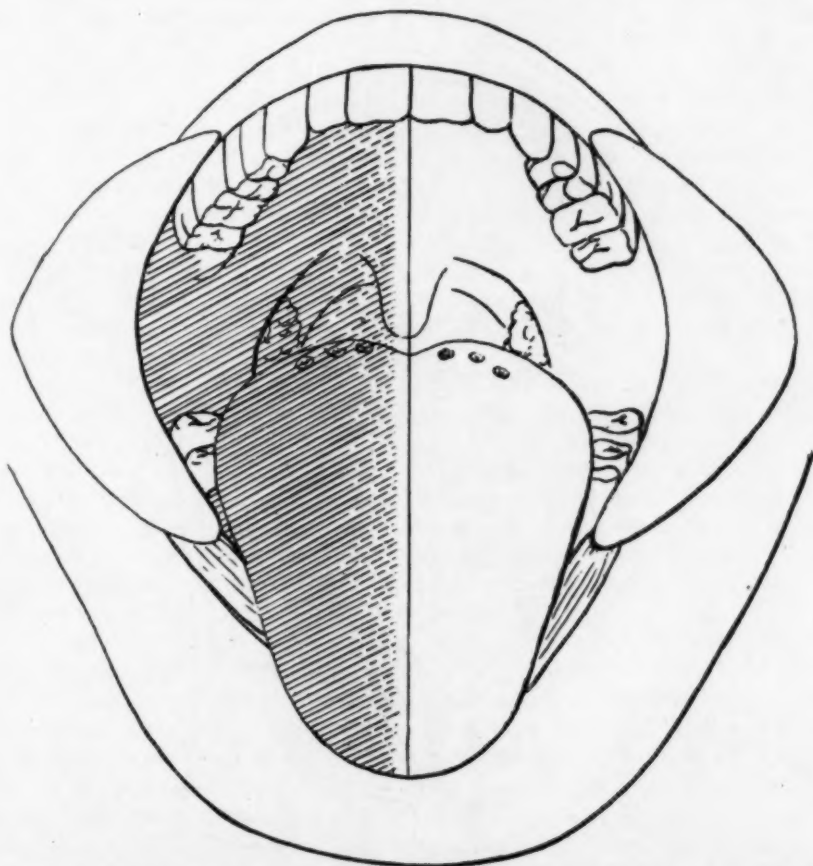


Fig. 1.—The shaded area shows the distribution of anesthesia to touch, pain and temperature over the tongue, hard and soft palates, uvula, tonsil and posterior pharyngeal wall on the right in Case 1, following the second operation.

right trigeminal nerve, over the mucous membrane of the right half of the buccal cavity, the right half of the alveolar processes, the tongue and hard palate and the anterior half of the right nasal cavity. Touch and pain are normally perceived over the entire surface of both external auditory canals, the external aspect of both tympanic membranes and both auricles. There is complete anesthesia to pain, temperature and tactile sensations over the entire right half of the inferior aspect of the soft palate. On its upper aspect the right

half of the soft palate is completely anesthetic for 1.5 cm. anterior to its posterior border. All of the portion of the right half of the pharynx lying between the level of the hard palate above and the pyriform fossa below is completely anesthetic to pain, temperature and tactile stimulation. Pain, temperature and touch are normally appreciated over the posterior half of the nasal cavity, and all of the nasopharynx above the level of a line drawn through the hard palate

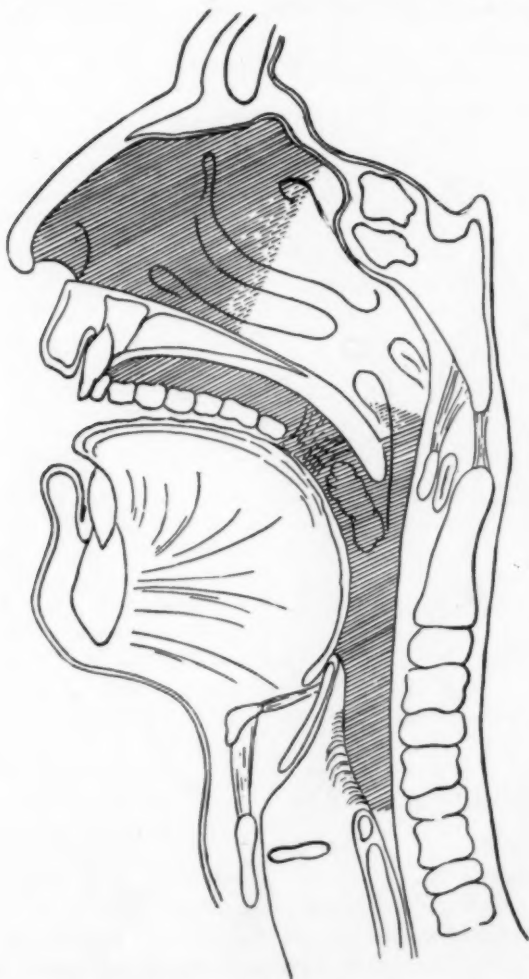


Fig. 2.—The shaded area shows distribution of anesthesia to touch, pain and temperature over the lateral wall of the nasal cavity, hard and soft palates and pharynx on the right in Case 1, following the second operation.

(Figs. 1 and 2). Sensation is normal over the epiglottis and the upper aspect of the larynx. Taste is absent for salt, sweet, bitter and sour over the entire right half of the tongue. There is paresis, Grade 2 to 3, of the right masseter and temporal muscles; the right pterygoid muscles are normal. The right

half of the soft palate, and the right superior, middle and inferior constrictors of the pharynx are totally paralyzed. There is loss of tone of about Grade 2, without actual loss of power, in the right half of the tongue.

CASE 2.—History.—Mr. F. W. McC., aged 52 years, came to the Clinic, March 28, 1922, complaining of a dull throbbing pain in the region immediately anterior to the right external auditory meatus, associated with paroxysms of sharp, stinging pain in the right side of the throat and right ear. He smoked a pipe incessantly. He had been deaf in the right ear for twenty years due to suppurative otitis media in childhood. He had never had herpes. Three weeks before his visit to the Clinic he had had a severe "cold" which lasted one week. As this infection was clearing up he noticed a sense of distress in the right side of the throat which he attributed to the cold. Two or three days later a dull throbbing pain developed in the region anterior to the right ear. The same day he began to have occasional, sudden, sharp, paroxysmal, stinging pains in the right side of the throat and right ear, lasting from twenty to forty seconds. By the second night he was beside himself, owing to the steady bombardment of paroxysmal pains. He was taking from eight to ten quarter grain (0.016 gm.) tablets of morphin daily. The narcotic relieved him of the dull throbbing pain; but mild paroxysms continued. After severe paroxysms he had a burning sensation and marked tenderness over the tragus. Talking and swallowing often induced attacks, although at times they occurred spontaneously.

Examination.—The patient was pasty and sallow. The systolic blood pressure was 90, the diastolic was 70, and the pulse was 58. Vision was 6/12 in the right eye and 6/6 in the left. The pupils were contracted (morphin?); the reflexes were impaired but present. The media were clear; the fundi were negative. There was a large perforation in the right tympanic membrane, but no evidence of active otitis media. The Wassermann reaction was negative, as were the urinalysis, blood counts and stereoscopic roentgenograms of the head. Neurologic examination was negative with the exception of diminution in hearing in the right ear.

Diagnosis: As in the first case, the diagnosis was trifacial neuralgia involving the auriculotemporal branch of the right mandibular division.

Treatment and Course.—March 31, 1922, the third division of the right trigeminal nerve was injected without influencing the pain. A day later the auriculotemporal nerve was injected, but the pain was not affected.

CASE 3.—History.—Mrs. E. M., aged 57 years, came to the Clinic, May 2, 1922, complaining of pain in the throat. With the exception of typhoid at 15 and pneumonia at 18, she had never been seriously ill. She had had ptosis of the right eyelid as long as she could remember. She had been perfectly well until the summer of 1921, when she developed occasional mild, sharp, paroxysmal pain, lasting a second or two, originating in the right side of the throat and radiating to the region around the angle of the right mandible. Gradually the paroxysms increased in severity, but they never came in series. After recurring several times daily for six or eight weeks the pain ceased as mysteriously as it had begun. On April 10, 1922, after she had taken a drink of cold water the trouble she had had during the summer of 1921 recurred suddenly. Now, however, the paroxysms continued in series over a period of three or four minutes. On April 17, she had a recurrence of paroxysms; since then they had recurred in attacks about twice a week. During all the attacks

in series she had felt that she was choking. She did not become cyanotic or really dyspneic. During the examination she had numerous paroxysms of pain. Although severe, the pain was not agonizing.

Examination.—The patient was a well preserved woman, moderately developed and fairly well nourished. Her blood pressure on two occasions was systolic 195, and diastolic 120; her pulse was 76. The blood Wassermann test was negative. The ear, nose and throat examinations were negative. Vision was 6/12 and 6/6 in the right and left eyes, respectively (with correction). The right pupil was slightly larger than the left; the reflexes were normal. Except for slight reduction in the caliber of the retinal arteries, the fundi were negative. Neurologic examination was negative, except for slight reduction in hearing in the left ear and ptosis, Grade 1 to 2, of the right upper eyelid.

Unfortunately, this patient left the Clinic before her examination was completed.

CASE 4.—History.—Mr. J. H., aged 61 years, came to the Clinic, April 10, 1922, complaining of pain in the region of the left external auditory meatus. As a child he had had "spotted fever" which resulted in the loss of the right eye. During adolescence he had had repeated attacks of suppurative otitis media; he had had a discharge from the left ear for ten years. From childhood until the age of 50, he had suffered from migraine. Nine years before he had become aware of repeated twinges of pain in the region immediately inferior and posterior to the left external auditory meatus. At first it was slight to moderate in severity; within thirty-six or forty-eight hours it became excruciating, and the attacks recurred at intervals of ten to thirty minutes. After eight to ten days the seizures diminished in frequency and by degrees ceased. Six years before he had a second attack lasting eight or ten days. Morphine alone gave relief. His third attack had occurred four years before and had lasted two or three weeks. The pain was not so severe or frequent as it had been. For a period of two weeks, commencing in the latter part of January, 1922, and ending in February, exceedingly severe attacks occurred at short intervals. His last attack commenced May 21, 1922, and was in progress at the time of examination. The pain was sharp, stabbing, and stinging; it came on suddenly in the area posterior and inferior to the left external auditory meatus, radiating down to the region of the angle of the left mandible. At onset the pain lasted only two or three seconds; in the fourth and last attack it persisted from thirty to fifty seconds. The pains never occurred in series of more than two or three paroxysms. Sudden movements of the head and neck, talking, drinking and fright seemed to induce paroxysms. Swallowing, especially of hot or cold fluids, was most likely to start the pain. A definite trigger area was not found.

Examination.—Physical examination revealed a man old beyond his years. He had a glass eye on the right, severe pyorrhea, systolic blood pressure 220 and diastolic 110, pulse 72. He had arteriosclerosis, Grade 3. The Wassermann reaction was normal. Vision of the left eye was 6/20 (with correction). The pupil was small and irregular; the reflexes were normal. The field was normal to rough test. The iris was atrophic; the globe was rather soft; and the pupil did not dilate. The fundus was seen with difficulty; there were no definite vascular changes. Ear, nose and throat examination revealed atrophic tonsils, a normal nasopharynx and larynx, a glazed appearance of the surface of the mucous membrane of the pharynx and slight irregularity of the nasal septum. When inspissated cerumen had been removed from the right external

auditory canal, the tympanic membrane was found thickened. The left external canal was filled in its internal two-thirds with fibrous and mucous polyps and mucopurulent exudate. Stereoscopic roentgenograms of the left mastoid showed cloudiness, Grade 3, while three biopsies on the polyps proved them to be myxomatous. The Bárány examination showed loss of function of the left vertical canal.

Treatment and Course.—As the patient was not a good operative risk the polyps were removed, and radical operation deferred.

Following the return of pain, May 21, the patient was seen in the neurologic section where the only additional findings on complete examination were static and movement tremors of moderate severity, and slight incoordination of the extremities. There was no loss of sensation to touch, pain, or temperature in the tongue, pharynx, hypopharynx, nasopharynx, soft palate, pillars, tonsils, and external auditory canals. Taste was preserved anteriorly and posteriorly over both sides of the tongue. During examination, May 24, he had a sudden attack of excruciatingly severe pain lasting about thirty seconds; it appeared and disappeared suddenly. During the paroxysm he coughed in a hacking manner every two or three seconds. Afterward he continued to hack, though less often, to relieve a sense of irritation in the left lateral pharyngeal region.

DISCUSSION

The distribution of pain in the first two cases was considered atypical, but the paroxysms were exactly like those of trifacial neuralgia. Following the recurrence of pain after section of the sensory root of the gasserian ganglion in the first patient, and after unsuccessful alcohol injections in the second patient, it became apparent that the condition was similar to trifacial neuralgia, but that some nerve other than the trigeminal was involved. Harris' discussion of glossopharyngeal neuralgia convinced me that the patients were suffering from that disturbance.

Two of the patients had hypertension (Cases 3 and 4) and one (Case 4) also had active disease of the middle ear. The second patient had no active disease, but had been deaf for twenty years as a result of suppurative otitis media in boyhood. The first patient had no such complicating factors, yet his paroxysms were more severe than those of the other three patients. No relationship could be established between these complications and the attacks of paroxysmal pain.

In the third and fourth patients, in addition to the paroxysms there were symptoms ordinarily interpreted as suggestive of irritation of the vagus. The third patient had a choking sensation with the attacks; the fourth coughed in a hacking manner throughout most of the paroxysms. In future studies attempts will be made to clarify this and other moot points. The possibility of the condition actually being vagoglossopharyngeal neuralgia must be considered.

The absence of taste following evulsion of the glossopharyngeal nerve and section of the pharyngeal branch of the tenth nerve brings

up the matter of the distribution of taste fibers. Luciani¹³ believes that the theory advanced by Carl,¹⁴ von Urbantschitsch,¹⁵ and first stated by Panizza¹⁶ in 1834, is the most acceptable. According to these authorities, taste fibers are derived entirely from the ninth nerve. The chorda tympani is composed of secretory and vasodilator fibers only. Unfortunately taste and ordinary sensation were not tested in the first patient prior to either of the operations. Hence, while the evidence is sug-

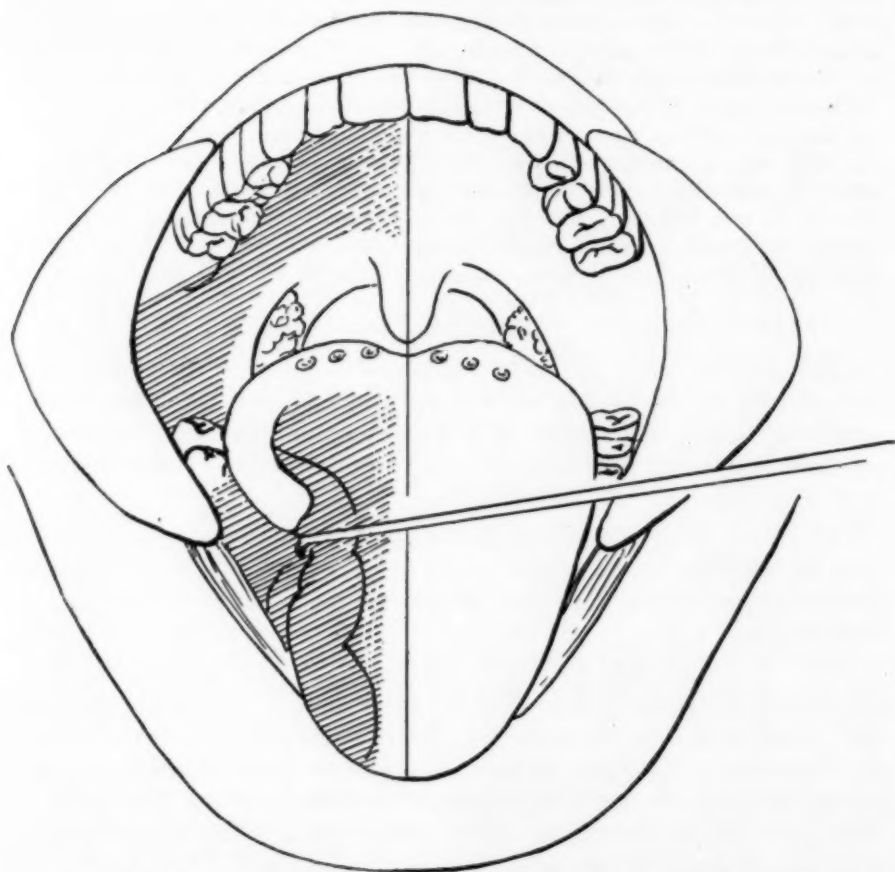


Fig. 3.—The shaded area shows the distribution of postoperative anesthesia to touch, pain and temperature over the tongue, hard palate and anterior part of the soft palate on the right in a case of trifacial neuralgia.

gestive, it is not conclusive that the ninth nerve subserves taste to the tongue in its entire extent.

13. Luciani, L.: *Human Physiology*, translated by F. A. Welby, London, Macmillan Co. 3:401-405, 1915.

14. Carl, quoted by Luciani.

15. Von Urbantschitsch, quoted by Luciani.

16. Panizza, quoted by Luciani.

The preservation of sensation to tactile, pain and thermal stimulation in the posterior part of the right nasal cavity and the right half of the nasopharynx prompted an investigation of the sensory changes in the nasal, pharyngeal and buccal cavities in a patient in whom section of the sensory root of the gasserian ganglion for trifacial neuralgia had been performed.

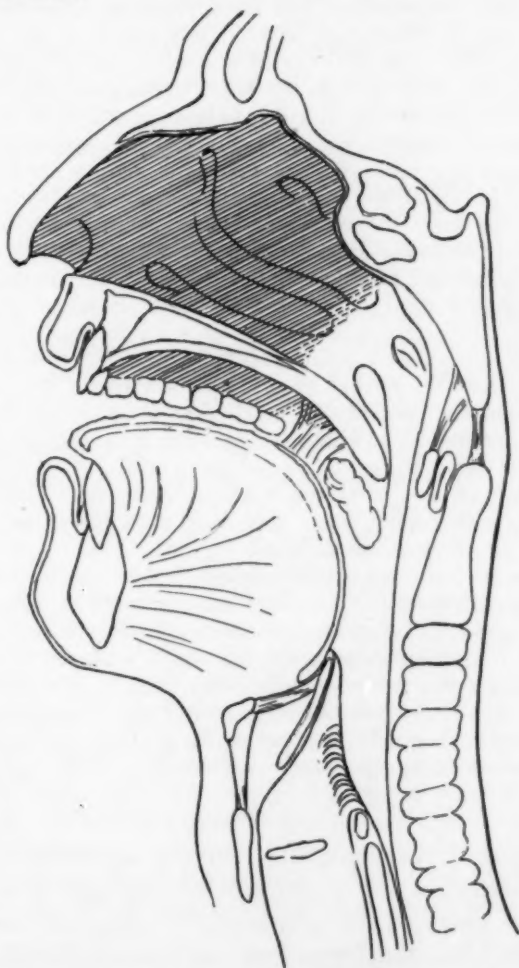


Fig. 4.—The shaded area shows the distribution of postoperative anesthesia to touch, pain and temperature over the lateral wall of the nasal cavity, hard palate and anterior part of the soft palate on the right in a case of trifacial neuralgia.

REPORT OF CASE OF TRIFACIAL NEURALGIA

CASE 5.—Miss J. L., aged 42 years, came to the Clinic, May 2, 1922, complaining of paroxysmal, stabbing pain. The attacks lasted four or five months and had occurred every one or two years for seventeen years. The pain always started in the region of the mental foramen of the right mandible and darted posteriorly and upward.

On May 13, 1922, the posterior root of the gasserian ganglion was cut, the motor branch being preserved.

Examination revealed total anesthesia to tactile, pain, and thermal stimulation over the cutaneous distribution of the right trigeminal nerve. Pressure sensation over this area was about —3. The anesthetic fields in the nasal and buccal cavities are well illustrated in Figures 3 and 4.

According to Schafer and Symington,¹⁷ the sphenopalatine ganglion derives sensory fibers from the maxillary and glossopharyngeal nerves, and from the geniculate ganglion of the seventh nerve. Only a few of the fibers from the maxillary nerve enter the ganglion, most of them being distributed directly to the nose and palate. The nerve to the nasopharynx originates in the back of the ganglion, often in common with the Vidian nerve.

In consideration of these data it is clear that the nasopharynx and a variable portion of the posterior part of the nasal cavity receive their afferent fibers from a source other than the trigeminal nerve, and the trunk of the ninth. At the time of operation the glossopharyngeal nerve was evulsed from the jugular foramen. The actual rupture of the nerve occurred at the lower end of the petrous ganglion. For this reason it cannot be determined with any degree of certainty whether or not the tympanic branch of the ninth nerve was interrupted in its course. This leaves open the question whether the afferent fibers of the sphenopalatine ganglion concerned with the innervation of the nasopharynx are derived from the intermediate nerve through the geniculate ganglion, the greater superficial petrosal nerve and the nerve to the pterygoid canal, or from the small branch from the tympanic plexus to the greater superficial petrosal nerve.

On account of the nature of the operative procedure it is impossible to draw conclusions with regard to the respective parts played by the ninth and tenth nerves in the pharynx and adjacent regions.

The operation performed was not considered ideal because section of both nerves was made peripheral to their ganglions. In order to relieve the patient, however, the procedure advised by Sicard and Robineau was carried out in a modified form. Sufficient reason for the ablation of the superior cervical sympathetic ganglion was not presented. Very little is known about the pain due to irritation of the sympathetic ganglions. Pain of sympathetic origin is not well localized and is generally of a thermal quality. As yet sufficient time has not elapsed to warrant conclusions on the efficacy of the operation.

CONCLUSIONS

Glossopharyngeal neuralgia is a definite clinical entity, differing from trifacial neuralgia only in the area of distribution of pain.

The afferent fibers of the sphenopalatine ganglion concerned with the innervation of the nasopharynx are not derived from the trigeminal nerve.

17. Schafer, E. A., and Symington, J.: *Quain's Anatomy*, New York, Longmans, Greene & Co. 3:Pt. 2, 21-23, 1909.

LESIONS IN THE BRAIN OF A PATIENT WITH POSTENCEPHALITIC PARALYSIS AGITANS*

J. CHARNLEY MCKINLEY, M.D., PH.D.

MINNEAPOLIS

The termination of certain cases of epidemic encephalitis in a state similar to paralysis agitans has been the subject of common observation and much comment, but our knowledge of the pathology of this type has been largely deductive. That the acute cases have shown the most marked lesions in the basal ganglions, midbrain, pons and medulla, has been interpreted largely in the light of the work of Hunt¹ who described progressive atrophy of the globus pallidus in a case of the juvenile form of paralysis agitans. It has thus been supposed that the symptomatology of postencephalitic parkinsonianism is based on degenerations in the corpus striatum, but observations on the pathologic anatomy have largely been lacking. So far as I have been able to determine, the reports of Foix² and Goldstein³ are the only ones dealing with this question from the objective point of view. The following case is reported as a contribution to this subject.⁴

REPORT OF CASE

History.—A farmer, 51 years of age, admitted to St. Mary's Hospital, Minneapolis, June 30, 1922, complaining of lethargy, was perfectly well until two years before when he had epidemic encephalitis. He was confined to bed for about three months in a state of lethargy. He gradually improved so that after four months he was able to do a small amount of light work. He has, however, been continuously lethargic since the first and has often fallen asleep at unusual times and in peculiar situations, though not while working. During the last few weeks the lethargy has grown much more pronounced so that now he sleeps almost continuously. Vision has been poor since his acute attack.

The family history was negative. The patient was in robust health up to the time of the present trouble. He had certain childhood infections, from all of which he recovered without sequelae. There was no history of operations or injuries. He had never had headaches or any disease of the eyes, ears,

* From the Neuropathology Laboratory, University of Minnesota Medical School, Minneapolis.

1. Hunt, J. R.: Progressive Atrophy of the Globus Pallidus, *Brain* **40**: 58, 1917.

2. Foix, C.: Les lésions anatomiques de la maladie de Parkinson, *Rev. Neurol.* **28**:593, 1921.

3. Goldstein, K.: Ueber anatomische Veränderungen (Atrophie der Substantia nigra) bei postencephalitischen Parkinsonismus, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **76**:627 (May) 1922.

4. This case was studied clinically by Dr. C. C. Tyrrell of Minneapolis, and his clinical findings are presented in this report.

nose or throat; he had had no respiratory diseases and he did not have influenza at the time of the pandemic. He had had no cardiovascular, gastro-intestinal or genito-urinary diseases. Venereal infection was denied.

Examination.—July 1, 1922, examination revealed that the patient slept and snored continuously. He was easily awakened and then answered questions intelligently, but he did not converse spontaneously. His facial expression was fixed and masklike. The skin was rough and dry. The pupils were equal, small and irregular and did not react to light. The eyes were usually fixed straight ahead. There was no ptosis. A small amount of pus was present in the left conjunctival sac. The ears, nose and throat were normal. There was marked pyorrhea alveolaris; the tongue was dry and coated. The neck, lungs and heart were normal. Sclerosis of the peripheral vessels was moderate. There was some tenderness in the right upper and lower quadrants of the abdomen with a little rigidity, but no muscle spasm. No masses were palpable within the abdomen.

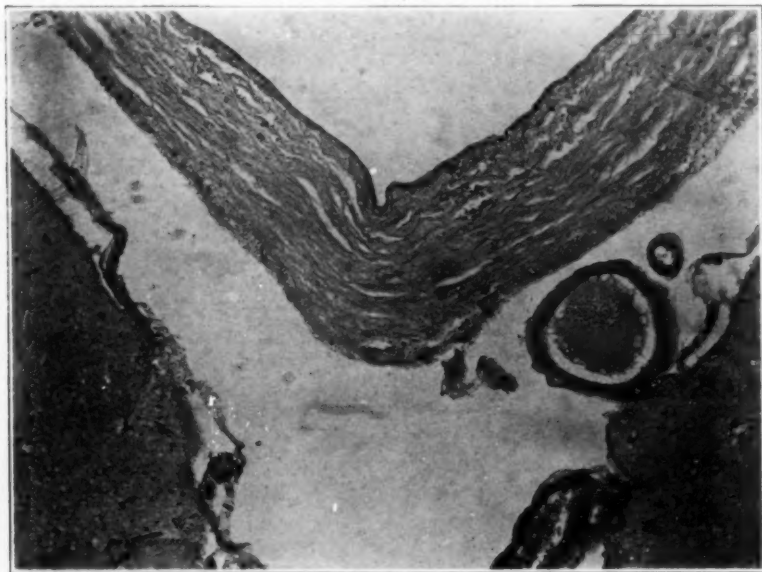


Fig. 1.—Fibrosis of the meninges over a sulcus in the region of the first left frontal gyrus. Hematoxylin and eosin; $\times 70$.

The upper extremities were continuously in a tremor indistinguishable from that of paralysis agitans. A "pill rolling" tremor of the fingers of the right hand was especially marked but was also present on the left side. The lower extremities were likewise tremulous and to such a degree that the constant rubbing had worn the skin and part of the flesh from the heels, leaving discharging sores. Spastic resistance was marked in all the skeletal muscles. The deep reflexes were all hyperactive but equal; there was no patellar or ankle clonus; the abdominal reflexes were active and equal; the plantar reflexes were normal.

The temperature (by axilla) was normal the first two days in the hospital, then varied between normal and 100 until the day of death, July 8, 1922, when

it rose to 103 F. The pulse ranged from 70 to 100 and the respiration from 20 to 25. The urine was normal. The blood examination showed a leukocytosis of 11,400. The blood Wassermann test was negative. No examination of the spinal fluid was made.

Clinical Diagnosis: Paralysis agitans following epidemic encephalitis.

Pathology.—Necropsy was performed eleven hours after death. Terminal bronchopneumonia, purulent bronchitis, chronic fibrous pleuritis and membranous adhesions about the ascending colon were found. There was no evidence of syphilis in the viscera or in the aorta.

The scalp and calvarium were normal. The dura was firmly attached to the pia-arachnoid by fine fibrous bands over the superior aspect of the brain, especially frontally and along the sides of the superior longitudinal sinus. On tearing the dura away, tags of these adhesions remained over the surface of the pia-arachnoid which were rather thick and opaque in some places, thin and transparent in others. The pacchionian bodies were distinct in their usual positions. The pia-arachnoid, especially over the frontal lobes, was thickened and

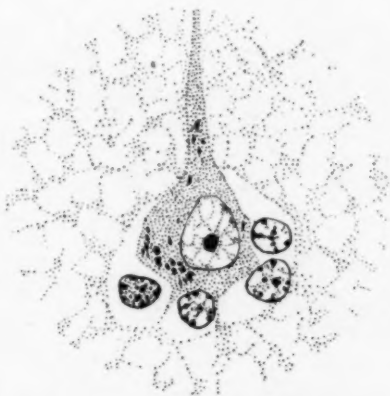


Fig. 2.—Camera lucida drawing of a cell in the left first frontal gyrus showing a slight amount of chromatolysis and four satellite cells. It is difficult to find many cells showing this amount of pathologic change in the cortex. Thionin; $\times 930$.

opaque, resembling at first glance a purulent exudate underlying a normal membrane. Gross section showed the thickening to be due to increase of fibrous connective tissue. It was especially marked along the vessels and over the sulci of the brain. There was some atrophy of the frontal lobes and a few adhesions on the under surface of the brain, especially marked anteriorly. The spinal cord was not removed.

The brain was put into 10 per cent liquor formaldehydi. Gross sections revealed no lesions visible to the naked eye.

Blocks of tissue were removed from various regions of the cortex, the corpus callosum, caudate and lenticular nuclei, thalamus, internal capsule, mid-brain, pons and medulla. The following strains were used: hematoxylin and eosin, thionin, polychrome methylene blue, Heidenhain's iron hematoxylin, Weigert's myelin sheath stain, Marchi's method for degenerating myelin sheaths

and Freeman's⁵ modification of the Warthin-Starry method for the demonstration of axis cylinders.

Sections from the cortex showed little abnormality except marked fibrous thickening of the pia-arachnoid, especially over the frontal lobes (Fig. 1). The thickening was due to simple fibrosis of the membrane without any evidence of an exudative inflammation. Tags of the torn adhesions which existed between the pia-arachnoid and the dura were seen microscopically as fibrotic projections from the superficial surface of the arachnoid. The cells, nerve fibers, interstitial tissues and blood vessels of the cortex were nearly normal throughout. There was some chromatolysis of the cortical neurons, not marked. Occasional cell bodies were found with three or four satellite glia cells closely applied to them (Fig. 2). There was no evident reduction in the number of cortical cells. Marchi and Weigert sections of the cortex showed normal myelin sheaths. No degenerating axis cylinders were demonstrable with the Freeman stain. The glia

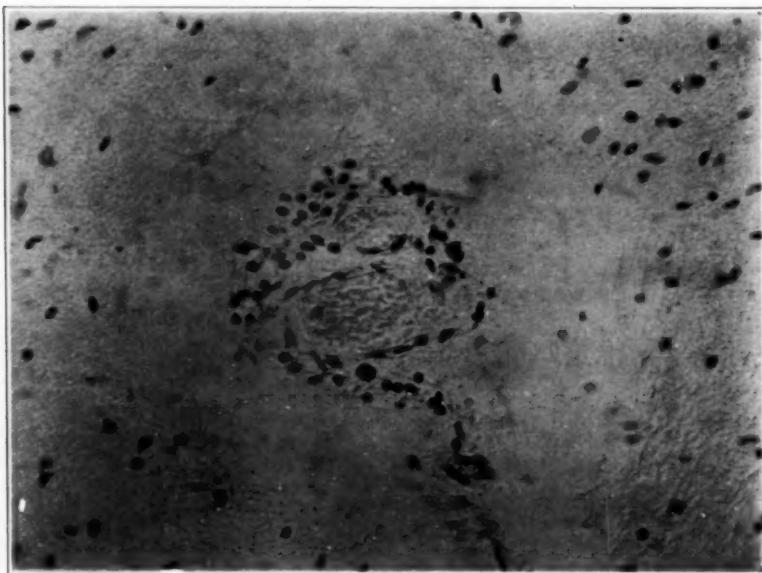


Fig. 3.—Photomicrograph of a perivascular collection of small round cells in the left globus pallidus. Only a few vessels in this region are affected to this extent. Thionin; $\times 300$.

showed no evidence of proliferation, either of cells or of fibers. Occasional small vessels were seen surrounded by a few cells of the lymphocyte and plasma cell type. Thickening of the vessel tunics was no more marked than one would expect in a person 51 years of age.

The globus pallidus, putamen and caudate nucleus have been studied with especial care because of the prominence they have been given by Hunt¹ in the case of paralysis agitans. The results are disappointing from his point of view. These regions contained insignificant evidences of disease as compared with the lesions found in persons who have died in the acute stages of epidemic encephal-

5. Freeman, W.: A Silver Diffusion Method for Staining Nerve Fibers in Paraffin Sections, *Arch. Neurol. & Psychiat.* 7:321 (March) 1922.

litis. With the exception of a moderate amount of chromatolysis and occasional examples of satellitosis, the cells of these regions were normal. There was no reduction in their number. The glia cells and fibers were not increased in the caudate nucleus or in the putamen. In the globus pallidus there was a slight increase in the number of glia cells in certain small areas, and a few cells resembling lymphocytes were present. Perivascular infiltration of small round cells was not observed in the putamen or in the caudate nucleus but was present, though by no means marked, in the globus pallidus (Fig. 3). The cells of the thalamus showed rather marked degenerative changes, largely in the form of chromatolysis, vacuolization of the cytoplasm, irregularity of the cell outlines and variations from normal in the position, size and shape of the nuclei. There was no evident reduction in the number of the thalamic neurons. Gliosis was more marked in this region than in the other parts of the basal ganglions, and perivascular collections of round cells were fairly frequent. Sections through

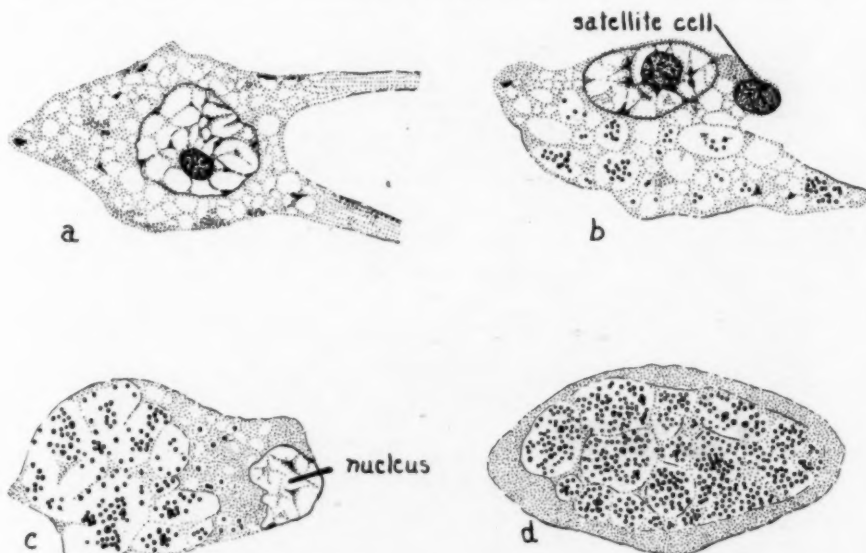


Fig. 4.—Types of degenerating cells in the nuclei of the oculomotor nerves. Compare with text. Camera lucida drawing; hematoxylin and eosin; $\times 840$.

the basal ganglions stained by the methods for studying axis cylinders and neurofibrillae and the myelin sheaths failed to reveal any degeneration of the nerve fibers. The internal capsules appeared normal. The ependymal lining of the third and lateral ventricles was normal.

Lesions were more pronounced in the mesencephalon than in any other situation. The nuclei of the oculomotor nerves were markedly affected. Practically all the cells of these nuclei were undergoing degeneration. The least injured were markedly chromatolytic with beginning vacuolization of the cytoplasm and irregularities in the shape of the nuclei (Fig. 4, a). From this type of cell every gradation could be traced through cells which were still more vacuolated with a few coarse yellowish pigment granules in the cytoplasm and in the vacuoles, and with more marked nuclear abnormalities (Fig. 4, b and c), to those cells which were only sacs containing considerable quantities of the

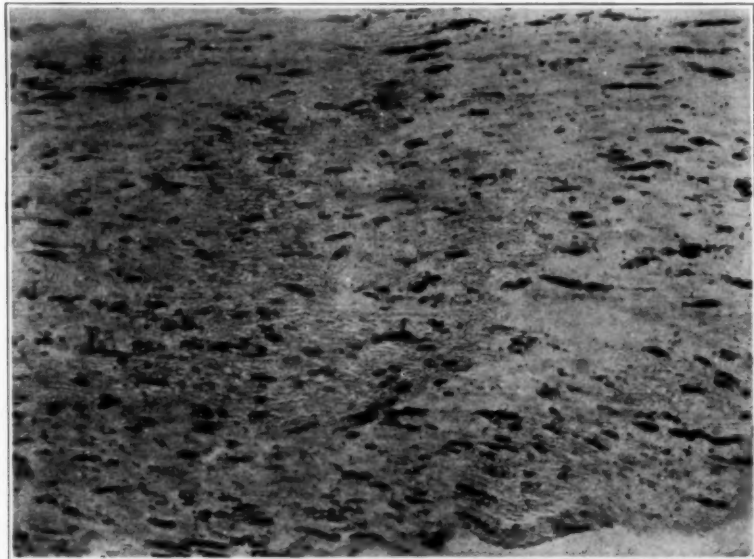


Fig. 5.—Degenerating myelin sheaths in the rootlet of the oculomotor nerve. Marchi's stain; $\times 80$.

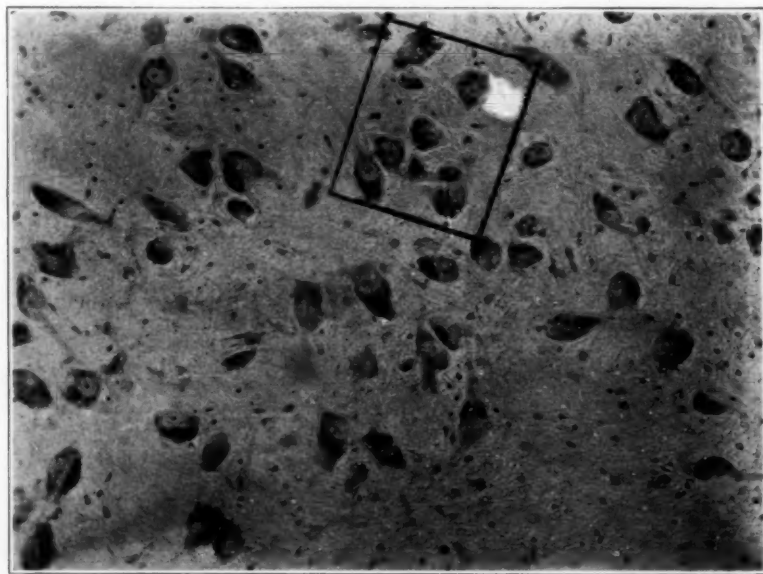


Fig. 6.—Photomicrograph of the cells in the substantia nigra of a woman 55 years of age, who died following a crushing injury of the thorax and pelvis in an automobile accident. Normal brain (N 345) for comparison. Polychrome methylene blue; 8 micron section; $\times 140$.

pigment granules (Fig. 4, *d*). Many of the cells had lost their processes altogether. Neuronophagia was not observed though an occasional satellite cell was seen (Fig. 4, *b*). The cells seemed merely to have undergone gradual disintegration without any chemotactic reaction. Parallel with the cell degeneration in the third nerve nuclei, sections of the third nerve rootlet stained with Marchi's, Weigert's and Freeman's stains all showed degeneration of a marked degree (Fig. 5). Increase in the number of glia cells was pronounced. Large clear glia nuclei were common and numerous elongated cells (Stäbchenzellen) were seen. Glia fiber proliferation was not marked. Perivascular infiltration of small round cells was common to most of the vessels, large and small, though the amount of infiltration was insignificant when compared with that in the acute cases. No abnormalities were observed in the colliculi. There were no marked lesions of the nucleus ruber. The ependyma of the aqueduct appeared normal.

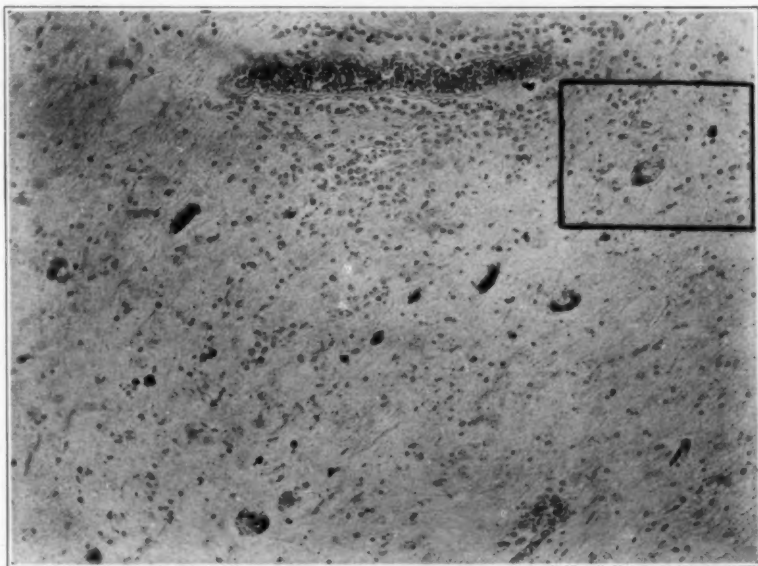


Fig. 7.—Photomicrograph of the substantia nigra in the case of post-encephalitic paralysis agitans. The loss of substantia nigra cells, the perivascular collections of round cells, the perivascular and diffuse gliosis, and the scattered pigment from broken-down cells are all evident when compared with the normal case (Fig. 6). Polychrome methylene blue; 8 micron section; $\times 140$.

The substantia nigra of either side was more profoundly altered than any other cell group. The cells were reduced to a small fraction of the normal number (Figs. 6 and 7). Those remaining were without exception chromatolytic; at least, this was the case in those cells in which the pigmentation was not so pronounced as to obscure the structure of the cytoplasm. No cells could be found as large as those of average size in normal brains used for comparison. The pigment in the cells appeared to be increased, but this was probably only relative, due to the shrinkage in the size of the cells which had crowded the pigment granules more closely together. Some of the cells contained little

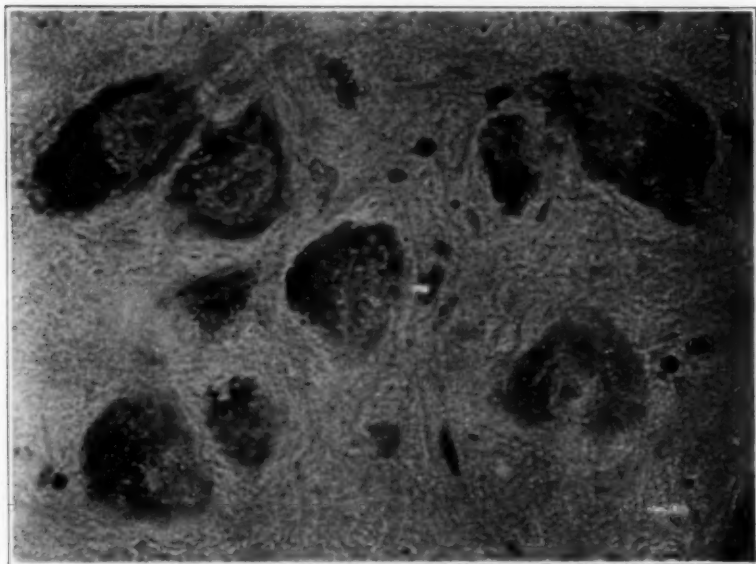


Fig. 8.—Photomicrograph of the field outlined in Figure 6. Normal substantia nigra cells for comparison with Figure 9; $\times 570$.

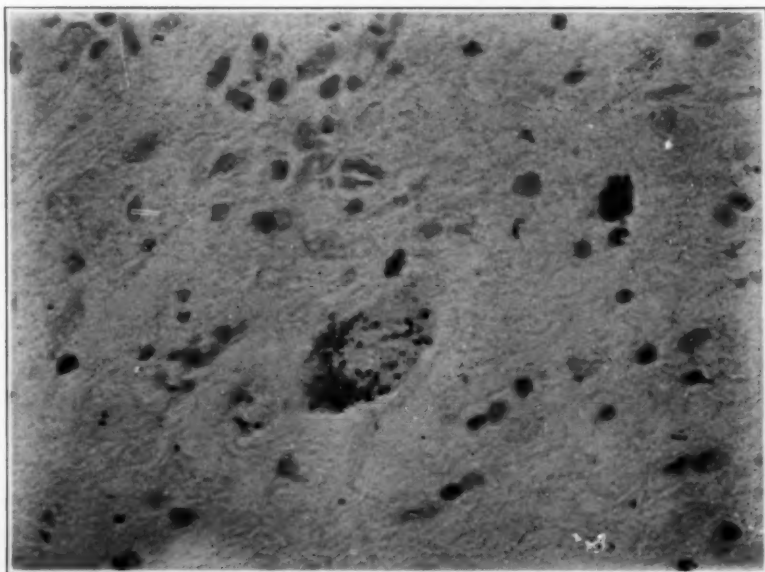


Fig. 9.—Photomicrograph of the field outlined in Figure 7. One of the surviving substantia nigra cells is shown. The scattered pigment from the previously degenerated cells and the gliosis are evident; $\times 570$.

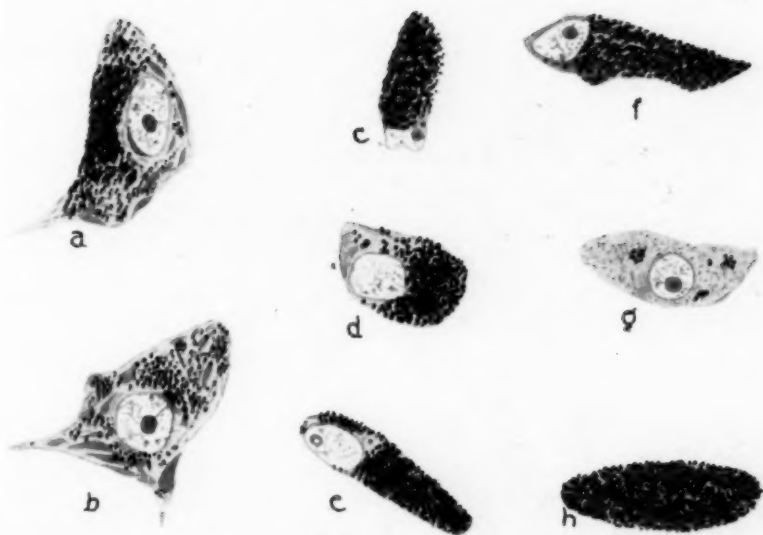


Fig. 10.—Camera lucida drawing of substantia nigra cells. Cells *a* and *b* are taken from the normal case (N 345). Cells *c* to *h* are from the case of post-encephalitic paralysis agitans. Atrophy of the cells, chromatolysis, nuclear abnormalities and relative increase in the intracellular pigment are evident in the postencephalitic case. The cells are all reproduced on the same scale of magnification from 8 micron sections stained with polychrome methylene blue; $\times 600$.

pigment; such cells were also found in normal cases. Decrease in size of the nuclei with marked eccentricity of the nuclear position was a regular finding. Neuronophagia did not occur in connection with these cells, and even examples of satellitosis were hard to find. Sections stained to demonstrate axis cylinders showed marked decrease in number of fibers; many of the remaining fibers were curled in the form of spirals and were irregularly swollen. That many cells had disappeared was evident, not only from their absence and from the absence of their processes, but also from the fact that within the boundaries of the substantia nigra there was more pigment scattered free in the interstitial network and in the glia cells than there was in the remaining nerve cells. This pigment was also found piled up along the walls of many of the vessels of the region, apparently in the process of being carried away along the perivascular spaces by migratory large mononuclear cells which had ingested

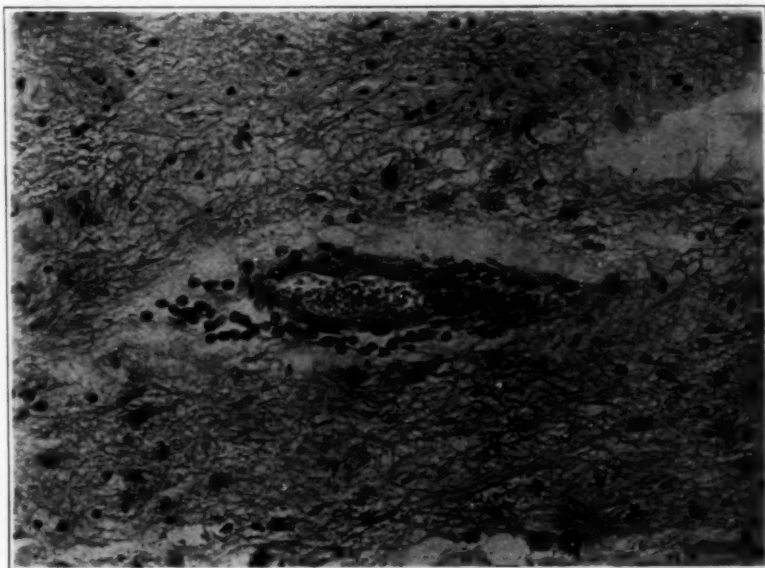


Fig. 11.—Perivascular infiltration of small round cells in the substantia nigra. A moderate spongy gliosis around the vessel and diffuse increase of glia cells are present. Hematoxylin and eosin; photomicrograph; $\times 240$.

it. Increase of glia cells was marked, but glia fiber proliferation did not appear to have kept pace with the cell increase. The gliosis followed closely the position of the substantia nigra, except for the region of the third nerve nuclei, as already noted. Perivascular infiltration of round cells (lymphocytes, plasma cells and large mononuclears, many of the latter containing an abundance of pigment granules) was more marked in this region than elsewhere. There were numerous fibroblasts scattered among the perivascular round cells; this is evidence of chronicity with a tendency toward healing which has not been noted in patients dying in the acute stages of epidemic encephalitis. The vessels were not any more sclerotic, however, than would be expected in a person of the patient's age.

The changes in the substantia nigra described in the foregoing are pictured fairly satisfactorily in the accompanying photomicrographs and drawings (Figs. 6-11); the captions are sufficiently descriptive to make it unnecessary to consider at this point the individual lesions illustrated.

Sections of the pons, medulla and the first cervical segment of the cord showed lesions of minimal degree. Chromatolysis was noted in many of the cells, but other cellular abnormalities were lacking. The occasional small areas of spongy gliosis may be interpreted as the end-results of the previous inflammation which undoubtedly involved this region during the acute stage of the disease. Perivascular collections of round cells occurred here, but they were far less extensive than in the midbrain. Some of the vessels were moderately sclerotic. The ependyma of these regions appeared normal. There were no lesions in sections from the cerebellum.

Weigert sections showed the same kind of thinning out of the myelin sheaths in patchy areas, especially in the midbrain and in the corpus callosum, as Spiller⁶ has recently described in the pons, medulla and cord in his case

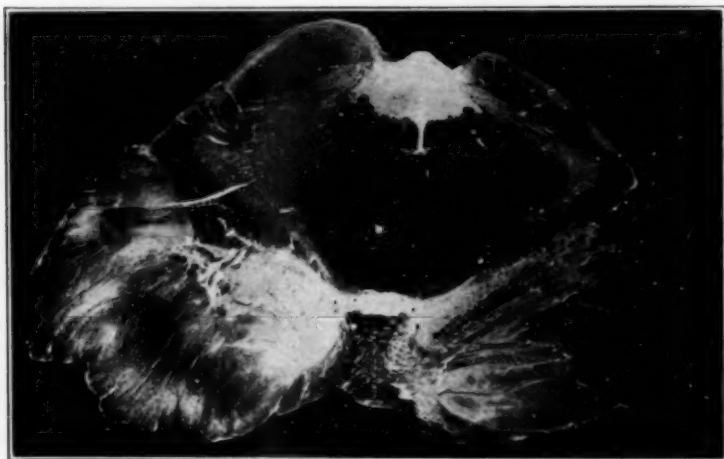


Fig. 12.—Photomicrograph of a Weigert preparation of the midbrain showing patchy areas in which the myelin sheaths are absent. The loss is especially marked in the peduncles; $\times 3$.

(Fig. 12). Corresponding degeneration of the axis cylinders does not appear to have occurred (except in the region of the substantia nigra, as already noted), so that the patches in this case reminded one very much of those seen in multiple sclerosis.

SUMMARY AND COMMENT

The patient had an attack of epidemic encephalitis with gradual recovery. He then developed the symptoms of paralysis agitans and entered on a stage of chronicity during which he became lethargic and

6. Spiller, W. G.: Epidemic Encephalitis with Myelitis, *Arch. Neurol. & Psychiat.* 7:739 (June) 1922.

finally died. The pathologic findings are in accord with such a progress of events, and confirm the opinion of von Economo,⁷ Globus and Strauss,⁸ and Schaller and Oliver,⁹ that epidemic encephalitis may become chronic with a lethal termination.

The principal interest in this case lies in the degeneration of the substantia nigra without extensive involvement of the lenticular nuclei, considering that the patient presented the typical clinical picture of Parkinson's disease. The case does not stand alone in this. Goldstein³ has described and pictured in a number of cases (he does not state how many) of postencephalitic parkinsonianism, lesions in the substantia nigra which are similar in every important respect to those here presented. Foix² has reported a similar case. He found:

"1. Massive lesions of the substantia nigra, discrete lesions of the lenticular nucleus, the optic thalamus, the tegmentum pontis and the colliculi."

"2. Persistence of fully active inflammatory lesions."

Foix has also studied the pathology in seven cases of classical paralysis agitans. In all of these he found abnormalities of the substantia nigra and of the lenticular nuclei, but the changes in the substantia nigra were constantly more intense than those of the lenticular region.

Souques and Trétiakoff¹⁰ describe the pathologic changes in the brains of three cases of paralysis agitans. In all of them degeneration of the substantia nigra was found and no appreciable lesions of the globus pallidus could be determined.

Souques¹¹ refers to some of Trétiakoff's work, the original report of which I cannot find, in which the latter studied nine cases of bilateral paralysis agitans and one unilateral case. In the nine bilateral cases he found bilateral lesions of the substantia nigra and in the unilateral case degeneration of the substantia nigra only on the side opposite the symptoms.

A fairly complete exposition of opposing opinions as to the pathology of paralysis agitans and the physiology of the basal ganglions and

7. Von Economo, C.: Ein Fall von chronischer schubweise verlaufender Encephalitis lethargica, München. med. Wchnschr. **66**:1311 (Nov. 14) 1919.

8. Globus, J. H., and Strauss, I.: Subacute Epidemic (Lethargic) Encephalitis, Arch. Neurol. & Psychiat. **8**:122 (Aug.) 1922.

9. Schaller, W. F., and Oliver, J.: Chronic Epidemic Encephalitis. Report of a Case: Clinical Record, Complete Necropsy and Detailed Histologic Study of the Central Nervous System, Arch. Neurol. & Psychiat. **8**:1 (July) 1922.

10. Souques, A., and Trétiakoff: Lésions du locus niger dans trois cas de paralysie agitante, Bull. et mém. Soc. méd. d. hôp. de Paris **36**:1027, 1920; abstr., Rev. Neurol. **28**:773, 1921.

11. Souques, A.: Rapport sur les syndromes parkinsoniens, Rev. neurol. **28**:534, 1921.

extrapyramidal motor system is presented in Souques' ¹¹ paper and in the discussion which follows it. Probably the cases with the paralysis agitans syndrome following epidemic encephalitis offer a better opportunity for settling the vexatious questions surrounding paralysis agitans and the functions of the various nuclei involved than the cases of paralysis agitans proper. At present it seems likely that lesions of the substantia nigra in relation to the parkinsonian syndrome will become more and more significant.

THE PATHOLOGIC FINDINGS IN THE HEART MUSCLE IN PROGRESSIVE MUSCULAR DYSTROPHY*

J. H. GLOBUS, M.D.

NEW YORK

While observing the clinical course of several cases of progressive muscular dystrophy, my attention was called to the manner in which patients who had remained in statu quo for fairly long periods of time, would, without warning, suddenly develop in rapid succession hypostatic pneumonia, pulmonary edema, hydropericardium and hydrothorax, culminating in death. Cardiovascular disturbance seemed best to explain these phenomena, and in two cases reported by Goodhart and myself,¹ lesions were found in the heart muscle, essentially of the same character as those found in the skeletal muscles.

In remarkably few cases of pseudohypertrophic muscular atrophy has a study of the heart muscle been made. Oppenheim,² referring to the few cases recorded,³ without giving reason for his opinion, says that he considers myopathic involvement of the heart muscle doubtful. Marinesco,³ in his splendid monograph on the disease of muscles, devotes a few paragraphs to the changes in the heart muscles occurring in the course of progressive muscular dystrophy. He says: "Among the muscles which offer an extraordinary resistance to the invasion by the pseudo-hypertrophic myopathy we must consider in the first place the cardiac muscle It is also true," he adds, "that publications in regard to this question are extremely rare, but this, perhaps, can be explained best by the fact that few investigators give sufficient attention to the microscopic examination of this organ. Nevertheless, Coste

* From the Department of Pathology of the Mount Sinai Hospital, New York City. This work was carried out during the tenure of the Sachs Fellowship in Neuropathology.

* Read before the New York Neurological Society, April, 1922.

1. Goodhart, S. P., and Globus, J. H.: On the Nature of Muscular Dystrophies with a Report on the Changes in Cardiac Muscle in Two Cases, *Neurol. Bull.* 1:387, 1918.

2. Oppenheim: Textbook of Nervous Diseases (translation by Alexander Bruce), Chicago, Chicago Medical Book Co., Ed. 5, 1911, p. 246.

3. Ross, James: On a Case of Pseudo-Hypertrophic Paralysis, *Brit. M. J.* 1:200 (Feb. 3) 1883. Hammond: Cited by Oppenheim and Marinesco. (Original article not available.) Stembo: Nimmt das Herz an der Pseudo-hypertrophie in den Fällen von Muskel-pseudo-hypertrophie? (Demonstration before Medical Association Vilna Nov. 12, 1897.) Cited by Oppenheim and Marinesco. (Original article not available.) Marinesco, G.: *Maladies des Muscles*, *Nouv. traite de méd. et de thérapeutique*, 1910, pp. 34 and 110.

and Cioja,⁴ Rinicker,⁵ Goetz⁶ and Hammond³ have observed hypertrophy of the heart in pseudohypertrophic paralysis without determining whether it was inherent in the disease or a coincidental complication. Mme. Sacara⁷ has noted, in four cases, irregularities in rhythm and intensity of the heart beat, while Jacobowitz has noted marked retardation of the pulse in one case. Stembo,³ employing the radio-

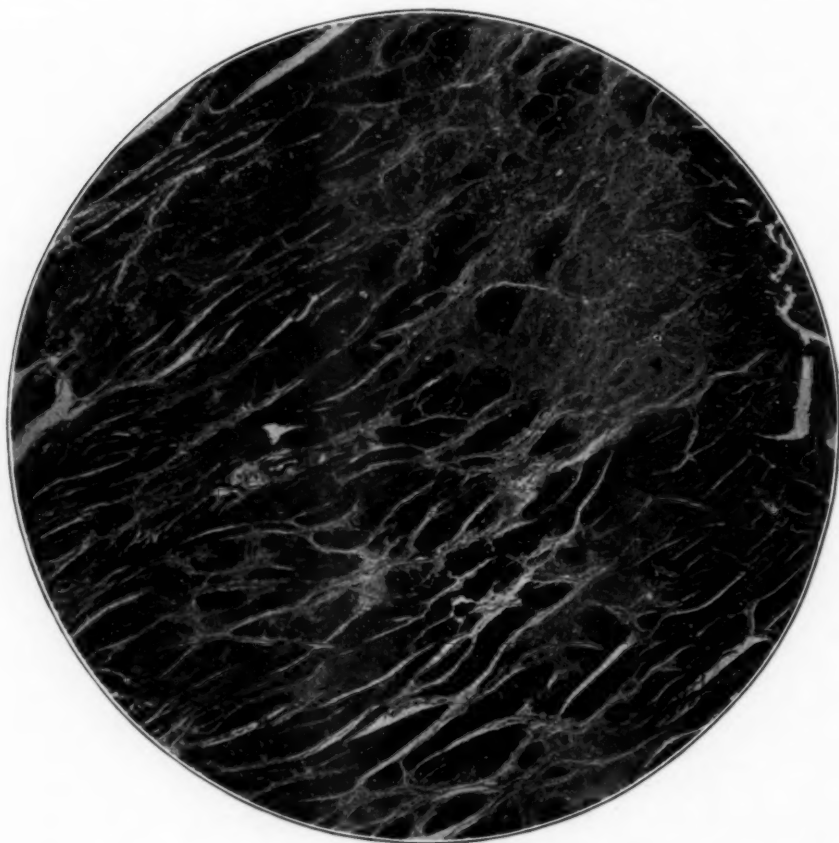


Fig. 1.—Section of the wall of the left ventricle showing a large typical fibrous scar with fairly conspicuous bundles of connective tissue radiating from it.

4. Original article not available.

5. Rinicker: *Verhandlungen der Physikalisch-medizinischen Gesellschaft in Würzburg*, 1860, p. 15. Rinicker's case of progressive muscular dystrophy, in a boy of 8½ years of age, showed enlargement of the heart during life. No anatomic findings are reported.

6. Goetz: *Beitrag zur Atrophie Muscularum Lipomatosa*, *Aertz. Intelligenz Bl.*, München 1879, p. 419.

7. Sacara-Tulbove: *Contribution à l'étude clinique de la paralysie pseudo-hypertrophique*, *Rev. méd.* **14**: 1894.

scope, was convinced that the heart in his case was affected by the disease, as it showed evidence of hypertrophy. Laignel-Lavastine⁸ described cardiac palpitation without valvular lesion as a rare complication of a case of primary myopathy of the Landouzy-Déjerine type.

In the chapter on the pathologic anatomy of muscular dystrophy, Marinesco⁸ describes a case in which the heart appeared involved and

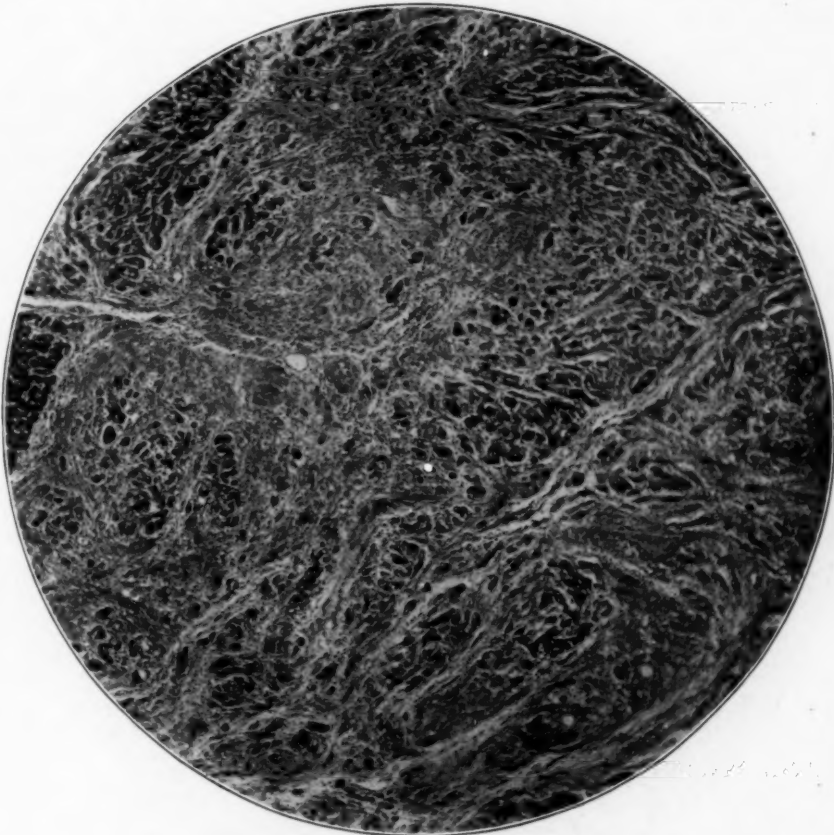


Fig. 2.—Section through a large scar in the wall of the left ventricle showing a marked increase in the interstitial connective tissue with a few small bundles of muscle fibers isolated in the scar.

showed typical lesions. He says, "In the study of the symptomatology of the primary myopathies one notes that the cardiac troubles do not generally participate in the general ensemble of characteristic phenomena of this disease, though the occurrence of myocardial disturbances has already been indicated." He adds that in the case of a child who died

8. Laignel-Lavastine: *Myopathie primitive progressive*, Arch. gen. de méd., n. s. 5:185, 1901.

of pulmonary tuberculosis complicating pseudomuscular hypertrophy, he found definite lesions in the cardiac muscle fibers and multiplication of their nuclei with increase of the interstitial tissue. Thus it appears that Marinesco inclines toward the belief that the cardiac muscle, in spite of its peculiar resistance to the disease, may be involved in the myopathic process.

Eulenberg and Cohnheim⁹ reported the heart in one case as normal in size and well contracted but of a rose-violet color. Charcot¹⁰ found

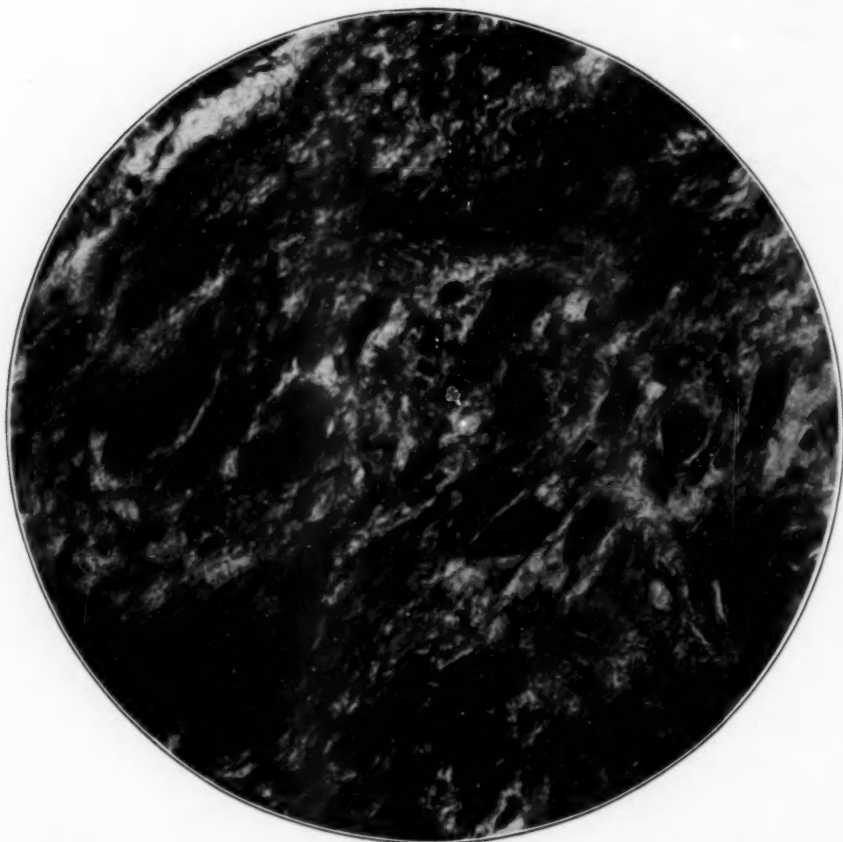


Fig. 3.—Section showing fragmentation of cardiac muscle fibers and marked proliferation of the interstitial connective tissue.

the muscle of the left ventricle free from pathologic change. In one case, Middleton¹¹ found the heart to be rather small and pale, but

9. Eulenberg and Cohnheim: *Verhandlungen der Berliner Mediz. Gesellsch.* **1**:191, 1865-1866. Cited by Potter. (Original article not available.)

10. Charcot: *Arch. de physiol.* **10**:228, 1871-1872.

11. Middleton, G. S.: *On the Pathology of Pseudo-Hypertrophic Muscular Paralysis with Remarks on a So-Called Degeneration of the Nervous System*, *Glasgow M. J.* **22**:81, 1884.

there was no marked increase in fat deposit and no hyaline degeneration. There was moderate increase of interstitial connective tissue. Goetz⁶ reported enlargement and paleness of the heart and the occurrence of yellowish patches in the papillary muscle about 1 cm. deep.

Ross³ reported definite myocardial involvement in a boy 12 years of age, who suddenly developed diarrhea, vomiting and general prostration culminating in sudden death. The heart muscle was flabby, the ventricles distended, the right ventricle filled with blood, the left



Fig. 4.—Section through a papillary muscle in the wall of the left ventricle showing interruption in the course of the muscle fibers by advancing scars.

empty. The wall of the left ventricle was one half inch (1.27 cm.) thick; the muscle was pale yellow and easily torn. The auricular walls were covered with a layer of fat. The heart muscle showed many atrophied fibers, which in many places were separated by interstitial connective tissue, increased in amount. Cardarelli,¹² describ-

12. Cardarelli: *Policlinico* 3:453, 1896 (Med. Section).

ing the heart of a boy 15 years of age, mentions only abnormal increase in the subepicardial fat. In his case, a severe attack of diarrhea and prostration led to sudden decline and death. Sachs and Brooks¹³ conclude that in their case the cardiac involvement formed no part of the myopathy. While the myocardium showed "diffuse fibroid infiltration," "the muscle fibers were found everywhere in a very natural condition," with neither atrophy nor abnormal pigmentation. Bunting,¹⁴ in one

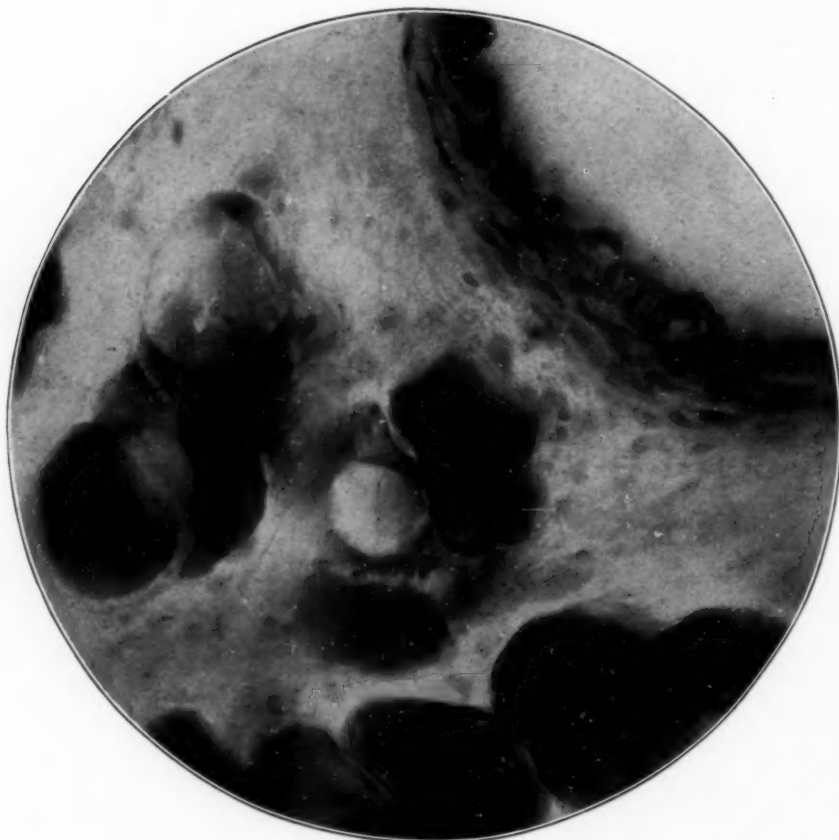


Fig. 5.—Section of left ventricle showing a vessel with the adventitia infiltrated with large masses of fat.

case, gave particular attention to involuntary muscle, striated (cardiac) and smooth (stomach). The heart was small and weighed 100 gm.; the valves were normal; the muscle was pale—grayish-pink. Sections

13. Sachs and Brooks: *Am. J. Med. Sc.* **122**:54, 1901.

14. Bunting, C. H.: *Chronic Fibrous Myocarditis in Progressive Muscular Dystrophy*, *Am. J. Med. Sc.* **125**:244, 1908.

of the wall of the left ventricle showed many translucent patches, irregularly distributed, which were most often found directly beneath the epicardium, with the subepicardial fat invading the muscle. The vessels were free from sclerotic changes. The lesions in the heart were identical with lesions in the skeletal muscle. He also reported increase in fibrous connective tissue and atrophy of muscle fibers in the wall of the stomach. More recently, Potter¹⁵ reported a case of muscular

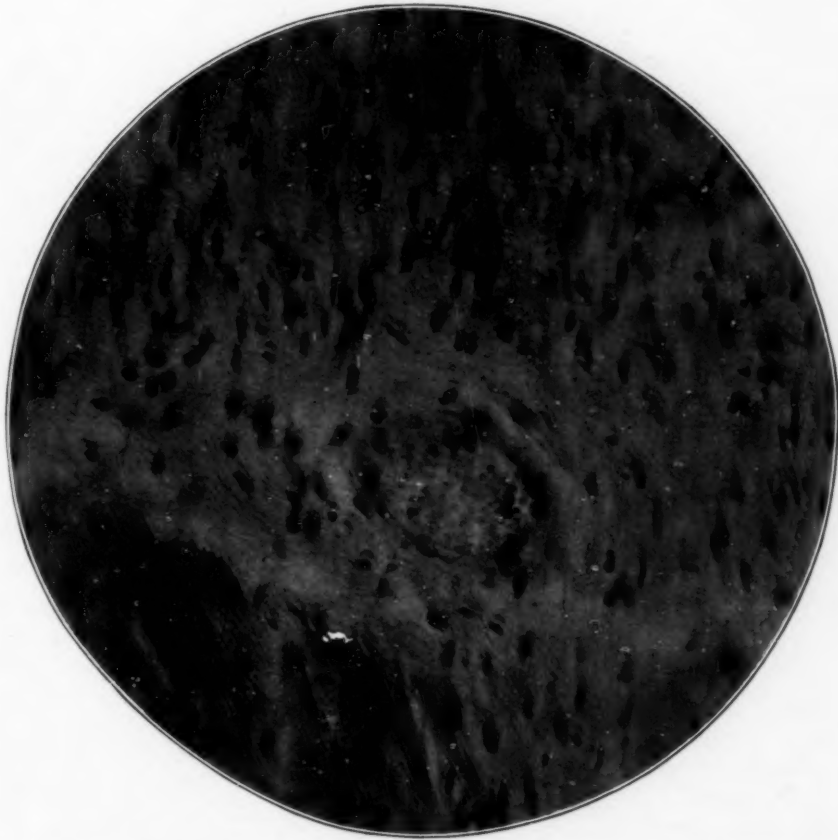


Fig. 6.—Section through the wall of the left ventricle showing a typical scar surrounded by a zone in which the muscle fibers show features typical of the skeletal muscles. There is also a distinct accumulation of fixed connective tissue cells about the blood vessel.

dystrophy with definite changes in the cardiac muscle, which he believed constituted a part of the myopathic disease.

15. Potter, F. C.: A Pathologic Study of a Case of Pseudo-Muscular Dystrophy with Change in the Heart Muscle, *New York M. J.*, Aug. 28, 1909.

REPORT OF A CASE

Clinical History.—A boy, 5 years of age, was examined by Dr. Roberto in 1916. His gait was unsteady. Calves and deltoids were well developed, but the pectoral muscles showed marked wasting. He had great difficulty in rising from the sitting posture and on rising from a recumbent posture had to climb up on his legs. A diagnosis of progressive muscular dystrophy was made. He gradually grew weaker and in June, 1919, it was noted that while there was no evidence of an infection, the pulse was rapid and variable, ranging from



Fig. 7.—Section through the wall of the left ventricle showing fragmentation and multiplication of nuclei and infiltration of the interstitial tissue by fibroblasts.

80 to 130. Frequent changes occurred without apparent cause. The arms were so weak that he had to be fed. In October, 1921, he was still weaker. Following the extraction of a tooth, there was collapse and rapid pulse. A large amount of acetone was found in the urine. There was continual vomiting, followed by a convulsion, and he died twenty-four hours after the operation.

Necropsy Findings.—The pectoralis major and minor, the deltoid, the intercostals, the diaphragm, the rectus abdominis and the obliquus externus and

internus (the muscles examined) showed advanced changes of progressive muscular dystrophy. The lungs were voluminous, quite edematous, the upper lobes fairly well aerated but the lower lobes heavy and hyperemic, with several small pneumonic areas. The heart was somewhat smaller than normal, the left ventricle well contracted and empty, the right dilated and filled with blood. The subepicardial fat was somewhat increased, and some fatty streakings were found under the endocardium. The heart muscle was pale, yellowish-red, flabby and friable. On section, the wall of the left ventricle showed many translucent

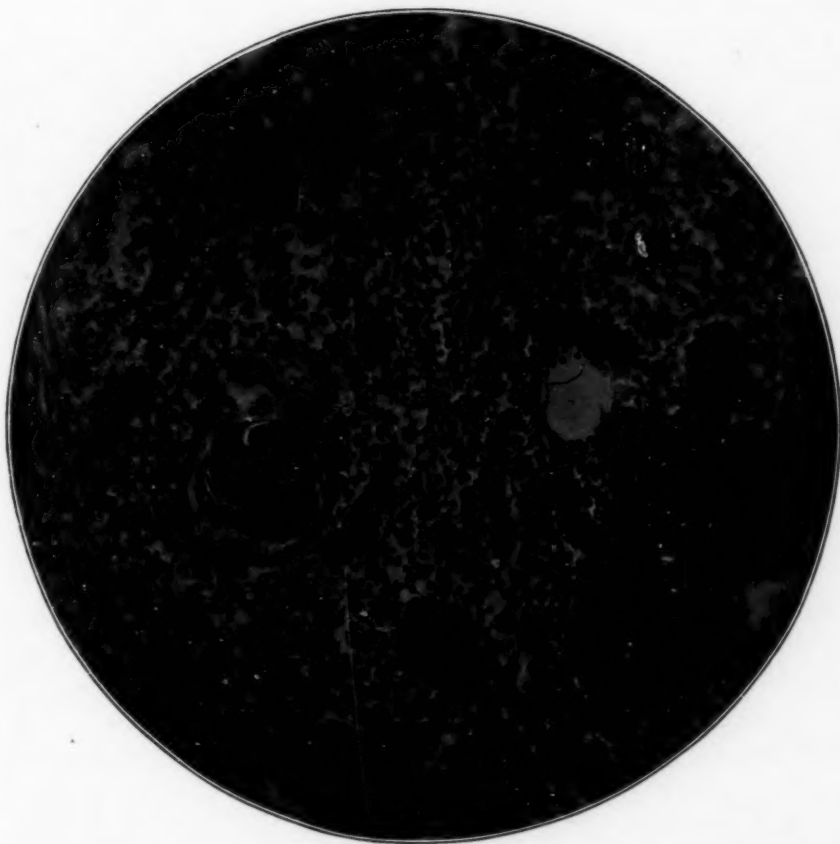


Fig. 8.—Section of the subinvolted thymus showing obliterating small blood vessels. They are in appearance not unlike Hassall's corpuscles.

patches, irregularly distributed. These patches bore no relation to the coronary or smaller vessels. The coronary vessels showed no pathologic change.

The thymus was large, covering almost the entire upper half of the pericardial sac. It was quite solid, about 1.5 cm. thick and rich in lymphoid tissue. The thyroid gland, although somewhat large, showed no gross pathologic change. The stomach and small intestines were normal. The large intestine was distended by solidified feces. The pancreas was normal, the liver markedly fatty, the kidneys congested, the testes and brain normal.

Microscopic Examination.—The liver showed marked fatty infiltration, diffusely and equally distributed. The pancreas, spleen, kidneys and testes were normal. The thyroid showed a simple struma.

Preparations from the wall of the left ventricle showed many connective tissue scars, varying in size (Fig. 1). They had not involved the perimysium externum, but were apparently produced mainly by the proliferation of connective tissue in the perimysium internum, leading to the isolation of individual muscle fibers (Fig. 2) and fragmentation of muscle fibers (Fig. 3). In younger, advancing scars small round fixed connective "tissue cells" were seen in large numbers (Fig. 4).

Fat infiltration was found in the connective tissue scars and about the blood vessels (Fig. 5), as it was in the skeletal muscles. As the degenerative process and the process of fibrosis were relatively mild in the heart, so the fat infiltration was rather meager. A larger amount of fat was under the epicardium, but this cannot be considered entirely pathologic.

Another feature, not unlike the pathologic change in the skeletal muscles, was the accumulation of many small round cells in the connective tissue trabeculae, in the fibrous scars, and, in a few instances, in the proximity of blood vessels (Fig. 6). This must not be interpreted as an inflammatory reaction but, as suggested by Marinesco and others, as a reactive phenomenon to the degenerative changes in the neighboring muscle fibers. Changes in the muscle fibers themselves were also observed. They partook mainly of the character of edema and swelling of some fibers, atrophy of others, with hyalinization and fragmentation (Fig. 7). Aschoff bodies were not found in the cardiac lesion described.

Findings in the thymus were extremely interesting. Particularly striking was the large number of vessels, some almost completely occluded, others partially so, by proliferation of the endothelial lining, while still others were open, but clogged with degenerating endothelial cells (Fig. 8).

COMMENT

Apparently this proliferation of the endothelium simulates the process of obliterating endarteritis, and when this process is complete, it gives rise to structures not unlike Hassall's corpuscles. The scarcity of these corpuscles, and the abundance of the transitional changes in the obliterating process of blood vessels, in the presence of subinvolution of the thymus, suggests a probable derivation of Hassall's corpuscles from obliterating vessels and that such a process might be responsible for the normal involution of the thymus.

The appended table was prepared to assist in determining the relation of the heart changes in progressive muscular dystrophy to a possible acute intercurrent disease, to some past illness, and to the pathologic process causing death. The table includes all cases which I have been able to find in the literature. All have shown fairly definite myocardial disease, though varying in degree. Although in three cases of the group a history of acute infection is given, the infection was insufficient to explain the diffuse nature and the morphologic character of the myo-

cardial involvement. In the column of intercurrent disease we find in one case pulmonary tuberculosis; in another, "a pulmonary condition," and in two others, pneumonia, while in the balance of the group pulmonary edema and grave gastro-intestinal disturbance were given as

RELATION OF HEART CHANGES TO DISEASE AS SHOWN IN THE LITERATURE

Investigator	Age of Patient, Years	Previous Illness	Nature of Terminal Illness	Anatomic Findings in Organs Other Than Heart	Changes in Heart Muscle
Goetz.....	10½	Scarlet fever	Not given	Congested lungs, spleen, kidneys	Enlargement of heart; increase of subendocardial fat
Ross.....	12	Pneumonia	Diarrhea, vomiting, prostration	No other findings *	Fibrous patches in wall of left ventricle; muscle pale, friable, fatty
Middleton.....	13½	Not given	A pulmonary condition	Pulmonary tuberculosis	Heart small, flabby, pale
Cardarelli.....	15	Not given	Diarrhea, prostration	Ulceration of large intestine	Enlargement; increase in subepicardial fat
Sachs and Brooks	12	Typhoid	Pneumonia	Bronchopneumonia, enlargement of spleen	Fibrous patches in walls of left ventricle; muscle pale, flabby, easily torn
Marinesco.....	Child Age ?	Not given	Pulmonary tuberculosis	Not given	Fibrous patches in the wall of the left ventricle
Goodhart and Globus	18	Not given	Pulmonary edema; hydrothorax; hydropericardium	Hypostatic pneumonia; hydrothorax; hydropericardium	Fibrous patches in the cardiac muscle; muscle pale and flabby; increase in fat; increase in the interstitial tissue
Goodhart and Globus	16	Not given	Pulmonary edema; hydrothorax; hydropericardium	Hypostatic pneumonia; hydrothorax; hydropericardium	Fibrous patches in the cardiac muscle; muscle pale and flabby; increase in fat; increase in the interstitial tissue
Bunting.....	15	Not given	Not given	Chronic suppurative bronchitis; bronchopneumonia; acute splenic tumors	Chronic fibrous myocarditis (translucent fibrous scars in heart muscle)
Potter.....	15	Measles, whooping cough	Bronchopneumonia	Fibrinous pleurisy; purulent bronchitis; subacute nephritis; passive congestion and fatty infiltration of the liver	Heart muscle pale, flabby; muscle fibers swollen, showing fatty infiltration and loss of striations

* Ross describes purulent fluid in pelvis of the kidney; the latter, however, being otherwise normal.

a cause of death. Here, again, there is little reason to suppose that the terminal acute pulmonary conditions or the gastro-intestinal disturbance accounted for the lesions of the heart, which were indeed of a chronic nature. Still, it is rather significant that pulmonary conditions should be found so frequently as a factor in the terminal issue of a large

number of cases. It is more reasonable to believe that the cardiac insufficiency was responsible for the development of conditions leading to death.

In studying the reports of the findings in organs other than the heart, we find few pathologic changes which could in any way suggest an etiologic factor responsible for the heart lesions other than primary myopathy. The character of the lesion offers further support to this assumption, for it appears that we are dealing with the same pathologic process in the heart as is found in the skeletal muscles. It is true that the process in the heart muscle is not so far advanced as in the striated voluntary type. This is probably due to a certain amount of resistance or partial immunity on the part of the heart muscle to the morbid process. This resistance or immunity prevents the heart muscle from going beyond the stage of fibrosis. However, the disease of the heart muscle must have advanced sufficiently in the early stage of the disease to permit its possible detection at that time by modern (electrocardiographic) methods. Such studies may hereafter yield very interesting results.

CONCLUSIONS

1. The heart muscle does not entirely escape the myopathic process characteristic of progressive muscular dystrophy.
2. It shows changes of a milder degree than those found in the skeletal muscles.
3. A few cases have been reported in which definite myocardial changes were found, but they have not always been looked upon as a part of the generalized process.
4. Probably myocardial changes will be more frequently found in progressive muscular dystrophy if the heart is systematically studied in every case.
5. The involvement of the heart muscle plays an important rôle in the terminal course of the disease and is perhaps responsible for some of the conditions which lead to rapid and fatal issue.

DISCUSSION

DR. WALTER TIMME: There are cases of muscular dystrophy in which there is marked myocarditis demonstrable in life, and many cases in which it is not demonstrable until after death. A clergyman of 82 years, who had had the disease since infancy and who had to be carried to the pulpit, had no sign of myocardial disease. The dystrophy was present in fourteen descendants in three generations; in none of them was there myocardial disturbance. A more rapid form of the disease, described by Gowers, causes death from the sixteenth to the nineteenth year and fatty degeneration of the heart assumes marked proportions. I think this disease is not exclusively a muscular one. It is not a

pure myopathy, but the myopathies are the most outstanding feature. Other atrophies, other dystrophies are seen, but not always. There may be only slight muscle changes. But necropsy will reveal disturbances in the anterior horn cells and in Clarke's column, in the anterior gray substance, in Lissauer's zone, which are not at all correlative to the slight muscular disturbance. In other cases at necropsy there are no spinal cord changes, but there have been marked muscular dystrophies. I roentgenograph all my patients for thymus; a large proportion have persistent thymus, and certain ones show a pineal shadow in early life. Marburg has published a paper on early pineal disturbance, accompanied by abnormal fat deposits about the body. I think we are beginning to see this disease as one in which the degenerative process is dependent on deficiency disturbances, probably in the control of some one or a group of the internal glandular units.

DR. FOSTER KENNEDY: Dr. Globus said that a certain number of the patients studied had died from a water logging of the chest. It would be interesting to know whether this had caused the death of his patient. I saw a child 13 years old, who died six years later with pseudodiaphragmatic paralysis. She suffered acute cardiac dilatation for three weeks before death, and had hydrothorax. The acute cardiac dilatation was not the primary lesion, but I thought it was a consequence of the heart strain.

DR. GLOBUS: I agree that progressive muscular dystrophy is a more or less generalized pathologic condition. In one case I found marked fatty infiltration in the liver, with no other pathologic changes in that organ. It was diffuse and bore no definite relation to the vascular or hepatic system of vessels and ducts. Bunting reported changes in the musculature of the large blood vessels. I noted hydrothorax and hydropericardium in three cases. The excess of pericardial and pleural fluids is best explained by the changes in the heart muscle.

DR. FOSTER KENNEDY: My experience has been that changes in the anterior horn and Clarke's column are not common. The neural changes occurring in paralysis are identical with those occurring after amputation, brought about by disuse. I think the changes in the spinal cord are secondary to those in the muscle. If we are to consider this as primarily an anterior horn lesion and a minor muscular disease, we become extraordinarily fogged in our classification of the pathology. Dr. Timme's experience has not been mine.

DR. WALTER TIMME: Three cases were reported by Kollarit and two by Schultz. These necropsy findings showed changes in the anterior horn cells in some cases, but they were not comparable to those in poliomyelitis. The cells were smaller than normal, but not degenerated. There was some disturbance of the fibers of the anterior cord substance. There were smaller cells in Clarke's column than normal. However, these findings are not universal. In cases of widespread muscular dystrophy there were often very few small cells. The conditions appeared to be absolutely independent of each other. It seemed to be a general atrophic condition involving the cord cells, as part of the general process and not as a secondary condition.

DR. GLOBUS: Modern neuropathologists have to account for two types of lesion: degeneration and inflammatory conditions. Poliomyelitis is a purely inflammatory lesion. It may have secondary degenerative processes. The cord changes in progressive muscular dystrophy are certainly not inflammatory. Are they to be considered as degenerative? Every degeneration in the central nervous system is accompanied by changes in the blood vessels and immediate

vicinity. The modern pathologist in such instances need not study the parenchyma; he studies mainly the relations of the glial elements. In degenerative lesions of the central nervous system one must find cells carrying away the products of destruction in the cord. The modification in size or shape of a nerve cell is not necessarily a pathologic phenomenon—it may be due to technical errors in preparation of material for study. But the glial reaction, accumulation of gitter and other phagocytic cells in the adventitial spaces is evidence of a degenerative process. Such changes are not encountered in the spinal cord in progressive muscular dystrophy.

SEX DEVELOPMENT AND BEHAVIOR IN MALE PATIENTS WITH DEMENTIA PRAECOX

CHARLES E. GIBBS, M.D.

Associate in Internal Medicine, Psychiatric Institute

WARD'S ISLAND, N. Y.

SEX DEVELOPMENT

The observations of Marro and others¹ have indicated the frequent occurrence of developmental defects of the reproductive apparatus and the physical characteristics of sex in idiots, cretins, epileptic persons, criminals, and persons with mental disease beginning at puberty. In male patients, varicocele, undescended testicle, hernia, hypospadias, unusually large and small testicles, deficiency of pubic hair and beard, and disturbance in skeletal growth, were reported to be more frequent than in controls. These observations were made before there was much evidence to indicate that certain endocrine glands are in some way essential to the normal development and functional activity of the reproductive apparatus.

More recently Emerson has reported the frequency of a type of imperfect physical development called "status lymphaticus" in alcoholic patients² and in patients with dementia praecox.³ He found this type in 22 per cent. of 1,000 alcoholic and drug cases in the alcoholic wards at Bellevue Hospital. Many of these inebriates were psychopathic. The incidence of status lymphaticus in the general medical wards was "less than a quarter of that found among alcoholics." He found the same type of physical make-up in 29 per cent. of 208 patients with dementia praecox from 16 to 50 years of age examined at the Manhattan State Hospital. Davis⁴ found the same type in 23.68 per cent. of 119 cases of war psychoneurosis, but in only 12.6 per cent. of 119 surgical cases in soldiers presenting no psychoneurotic features.

The essential features for the clinical recognition of "status lymphaticus" refer to the physical characteristics of sex. Yet 13 per cent. of the alcoholic persons classed by Emerson as normal had the feminine type of pubic hair, and in 24 per cent. the testicles were noted as small. This suggests the extent to which the opinion of the

1. Marro, A.: *La Puberté*, Paris, 1902. Hall, G. Stanley: *Adolescence*, New York, D. Appleton & Co., 1911, vol. 1, chap. 6.

2. Emerson, Haven: Status Lymphaticus in Adults, Its Clinical Diagnosis and Importance, *Arch. Int. Med.* **13**:169, 1914.

3. Emerson, Haven: Note on the Incidence of Status Lymphaticus in Dementia Praecox, *Arch. Int. Med.* **14**:881, 1914.

4. Davis, T. K.: Status Lymphaticus: Its Occurrence and Significance in War Neuroses, *Arch. Neurol. & Psychiat.* **2**:414 (Oct.) 1919.

examiner must enter into the classification of persons into physical types, especially when several features are considered which are not accurately defined or measured.

In the absence of well recognized and clear cut types of physical make-up, and in view of our still limited knowledge as to the exact relation between the various endocrine glands and the development of certain tissues and structures, it would seem better to study and classify these various physical features as separate units. Even in attempting to make a rough classification of individual parts, like the testicles, as to their development, the lack of a standard, the difficulty of exact measurement and the opinion of the examiner make accurate classification difficult. This is as true for controls as for the cases being studied.

TABLE 1.—SEX DEVELOPMENT IN DEMENTIA PRAECOX

Age on First Admission	Number of Cases	Size of Testes				Femine Pubic Hair, Percentage	Deficient Beard, Percentage	Scrotal Implantation, Percentage	Patients with Undescended Testis
		Patients with Both Testes Smaller than the Average, Percentage	Patients with One Testis Smaller than the Average, Percentage	Patients with Both Testes Larger than the Average, Percentage	Patients with One Testis Larger than the Average, Percentage				
16-20, under 21 when examined	47	6.3	6.3	14.8	6.3	12.7	61.7	27.6	3
16-20, 21 or over when examined	58	3.8	9.6	32.6	19.2	13.4	34.6	23.0	1
21-25	96	5.2	12.6	21.0	6.3	4.2	26.3	17.8	1
26-30	51	5.8	13.7	21.5	11.7	0.0	11.7	23.5	1
31-35	45	2.2	15.5	20.0	15.5	2.2	8.5	4.4	2
36-40	34	5.8	5.8	5.8	20.5	2.9	2.9	11.7	2

Case Material.—In Table 1 certain findings in 325 male patients with dementia praecox have been tabulated according to the age at the time of first admission to a hospital for mental disease. The first group of forty-seven patients, from 16 to 20 years on first admission, were examined before they were 21 years of age. Eleven were 20 at the time of examination and the others were under 20. The remaining 278 patients in Table 1 were considered as having chronic cases, and from one to twenty years had elapsed between their first admission and the time of examination. All were 21 or over at the time of examination. The age on first admission was unknown to the examiner at the time of examination, being determined later. The cases were unselected, and no previously recognized cases of endocrinopathy were included.

Size of Testes.—Testes have been classed as average, large or small, according to the examiner's conception of the average normal adult size. This conception was based on examinations of patients in the

wards of a general hospital and on a clay model having the dimensions given in Cunningham's anatomy. Those testicles which seemed by palpation to be definitely larger or smaller than the average were so classed, but no attempt was made to decide in each case whether the variation was sufficient to be called abnormal. The figures, therefore, merely indicate the percentage of cases showing variation from the average; they do not attempt to represent the percentage of cases showing abnormality. By this means, and with all examinations made by one observer, the error due to the examiner's opinion in each case is reduced to a minimum. This method affords a satisfactory working standard for comparing the size of the testes according to the age of the patient on first admission. The results are so arranged in Table 1.

The testes of patients first admitted between the ages of 16 and 20 compared favorably in gross size with those of patients first admitted between the ages of 21 and 40. Small testes were no more frequent in patients admitted at an early age than in those admitted later in life. Large testes were nearly as frequent in patients admitted and examined between 16 and 20 as in those admitted between 21 and 40, and were more frequent in those admitted between 16 and 20 but not examined until they were 21 or over. In explanation of the latter observation two possible factors may be considered, one or both of which may have been operative. First, in some cases with the psychosis developing during puberty there may have been an overgrowth of testicular tissue which was not completed until 21 or later. Second, there may have been an accumulation in the wards for chronic cases of patients with large testes. The prognostic significance of disturbances of sex development with regard to ability to leave the hospital and to rapidity of deterioration cannot be determined until consecutive early admissions have been followed for several years.

The majority of the patients examined had testicles which compared favorably in size with the normal, according to the examiner's conception of the normal average size. This was found to be true even for patients admitted and examined when they were between 16 and 20 years of age. In less than 5 per cent. of all patients both testes were noted as small. Typical infantilism was not encountered, but in a few cases both testes and penis were quite small and pubic hair almost totally absent. Typical infantilism has been observed in two patients first admitted before 16 years of age, but not included in the series. In several patients one testis was small while the other was large, and when one testis was undescended the other was usually as large or larger than the average.

In these examinations it was frequently observed, especially in young patients, that the testis felt unusually hard or firm or rubbery. In many such the epididymis and vas could be palpated only as a cord

firmly adherent along the border of the testis, so that it could not be separated out and palpated between the fingers. Many of these rather firm testes were not of the normal shape, but were nearly round or had a deficiency of tissue at the lower pole. A ridged or corrugated surface has been rather frequently observed. These findings suggest the possibility of a growth or overgrowth of tissue without proper differentiation, or an excess of connective tissue.

In view of the reports⁵ of failure of spermatogenesis and degenerative changes in the gonads in dementia praecox, it might be expected that testes smaller than the average would be rather frequent. Tiffany noted, however, that parenchymatous degenerative changes were accompanied by increased connective tissue even in young subjects, and that size and consistency cannot be taken as absolute criteria of the extent of degenerative changes. It might also be expected that patients with a long duration of the psychosis would show smaller testes than recent admissions with a short duration. In twenty-two patients between 16 and 40 years of age when first admitted, there were twenty-eight testes classed as small. Eleven were in patients who had been in the hospital more than ten years, and seventeen were in patients who had been in the hospital less than ten years. However, patients in whom degenerative changes would be most definite and rapid might not live long enough to be in the hospital ten years, or increase of connective tissue might compensate for parenchymatous shrinkage and so prevent decrease in gross size.

Feminine Type of Pubic Hair.—Three types of distribution of pubic hair have been observed: feminine, semifeminine and masculine. Only such cases were classified as feminine as showed the hair growth to be definitely limited to the pubic area with the distinct horizontal limitation and no hair extending along the midline toward the umbilicus. This type is shown in Figures 1 and 2. It is to be noted that the feminine type is an average growth limited to the feminine distribution rather than such a degree of deficiency as to limit it to this area. The comparatively few cases of definitely deficient pubic hair usually showed the masculine type on close examination. It was frequently observed, however, that with a good growth of the feminine type there was little or no hair on the scrotum and perineum.

5. Obregia, M.; Parhon, C., and Mechia, C.: Recherches sur les glands génitales testicules et ovaires dans la demence precoce, *L'Encéphale* 8:109 (Feb.) 1913. Todde: Ricerche sulla funzione e sulla struttura della ghiandola sessuali Maschili nelle Malattie Mentali, *Riv. sper. di freniat.* 40:233, 1914, cited in *Sex Organ Changes in Insanity*, editorial, *J. A. M. A.* 65:254, 1915. Mott, Frederick W.: Normal and Morbid Conditions of the Testes from Birth to Old Age in One Hundred Asylum and Hospital Cases, *Brit. M. J.* 2:655 (Nov. 22) 1919. Tiffany, W. J.: Pathological Changes of the Testes and Ovaries in Dementia Praecox, *New York State Hosp. Quart.* 6:159 (Feb.) 1921.

The pure feminine type occurred in 13 per cent. of patients first admitted between 16 and 20 years of age, and in only 2.6 per cent. of those first admitted between 21 and 40. The difference is quite definite, since the type was accurately defined. That the feminine type is not merely a phase of male development is indicated by its occurrence in 13.4 per cent. of those first admitted between 16 and 20, but who were



Fig. 1.—Patient with dementia praecox. Age on admission, 18; present age, 23; feminine pubic hair; deficient beard; no hair on the trunk; scrotal implant; testes average size; long thorax.

not examined until after 21. Their average age at the time of examination was 23 years.

The semifeminine type gave the impression of a primary feminine growth with the addition of a few hairs along the midline or arranged

in a whorl over the abdomen, the horizontal demarcation remaining distinct. This secondary growth was usually of much finer and shorter hair than that over the pubis. This semifeminine type was noted in about 11 per cent. of the patients examined, and was fairly evenly distributed through the age groups. Cases of this type are shown in Figures 3, 4 and 5.

Small testes were associated with the pure feminine type of pubic hair in only four of nineteen patients, and large testes occurred in four.

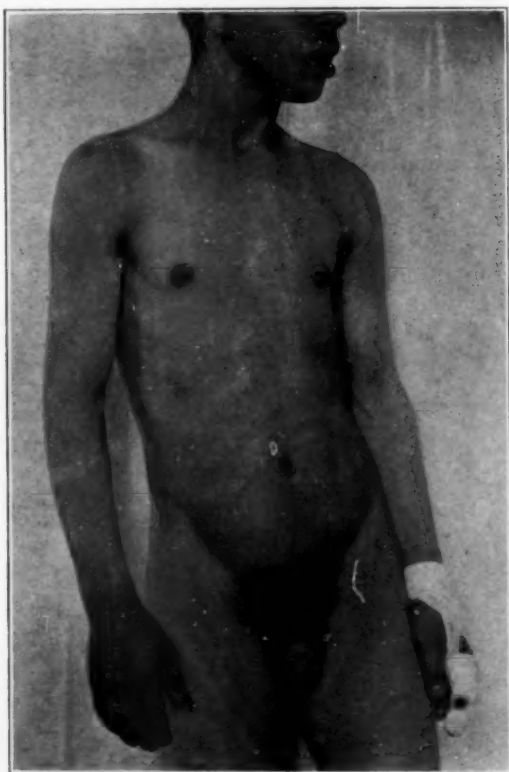


Fig. 2.—Patient with dementia praecox. Age on admission, 18; present age 23; feminine pubic hair; deficient beard; no hair on trunk; testes large; large bones and muscles.

In only two patients were both testes small. In two patients one testis was undescended, and in one of these the other testis was unusually large. Likewise in patients with definitely deficient pubic hair of the masculine type, there was no particular deficiency in the size of the testes.

Because of its persistence after puberty, the abundance of its growth and the size of the testes, it would seem that the greater frequency of

the feminine type of pubic hair in patients with early onset is something more than a retarded phase of normal male sex development. It seems more definitely a perversion or unevenness of development.

Deficient Beard.—Marro found absence of the beard in 13.9 per cent. of 395 criminals over 20 years of age, and in only one of sixty-three normal subjects. He also observed that absence of the beard was

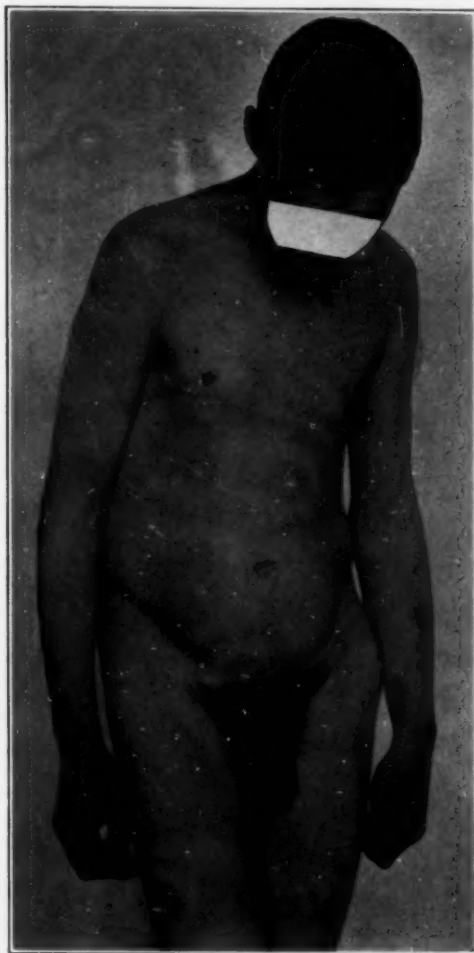


Fig. 3.—Patient with dementia praecox. Age on admission, 19; present age, 24; eunuchoid body contour; semifeminine pubic hair; testes large.

frequent in the insane, and quotes Clouston as having observed that rapid growth of the beard was a favorable prognostic sign in the insanity of adolescence.

In the patients tabulated in Table 1 as having a deficient beard, there was a total absence or marked deficiency of hair on the sides of

the face and jaw, under the chin and on the neck. The hair that grew over this area was present in small patches or tufts separated by much larger areas without hair. A distinction is to be made between the hair on the lip and chin and that on the jaws and neck. The latter is more characteristic of the male. The former is frequent in females after



Fig. 4.—Patient with dementia praecox. Age on admission, 16; present age, 21; semifeminine pubic hair; absence of beard; scrotal implant; testes large.

removal of the ovaries and after the childbearing period. Martin⁶ has designated the lip and chin hair as primary and that on the jaws as secondary. In Figure 6 is shown a two weeks' growth of beard which

6. Martin, Rudolph: *Lehrbuch der Anthropologie*, Jena, 1914, p. 373.

is confined to the lip and chin with no hair on the jaws and neck. This absence is also shown in Figures 2 and 4, which were also two weeks' growths.

From Table 1 it will be noted that deficient beard was quite frequent in patients admitted at an early age and gradually decreased in frequency in patients admitted at a later age. A scant growth of beard would be expected in the group examined during puberty, but it will be noted that 34.6 per cent. of those admitted early still showed the deficiency when they were 21 or over. In the group of patients between 21 and 25 years of age on first admission there were forty-six who



Fig. 5.—Patient with dementia praecox. Age on admission, 19; present age, 30; semifeminine pubic hair; scrotal implant; testes quite large; actively homosexual.

had been in the hospital long enough to reach the "chronic" wards. Ten of these forty-six, or 21 per cent., showed deficient beard after they had reached an average age of 25.8 years. Five of the ten were over 26.

Small testes were associated with deficient beard in only a few cases, being noted in only two of the eighteen patients with deficient beard in the 16 to 20 group examined when they were 21 or over. In ten of these eighteen patients, one or both testes were classed as large. Small testes were noted in only two of the ten patients with deficient beard in the 21 to 25 group who were examined after they had reached an average age of 25.8 years.

Scrotal Implant.—Patients were classed as having a scrotal implant when a fold of the scrotum on each side extended above and around the penis so that the penis seemed to emerge between these folds (Figs. 1, 4 and 5). Scrotal implant occurred rather frequently, as shown in Table 1, and was somewhat more frequent in patients admitted at an early age than in those admitted at a later age. This relation to age was not as definite, however, as that of the secondary sexual hair. This anomaly of genital development probably should not be classed as a secondary sex character in the same category with the secondary sexual hair and having the same relation to the gonads.



Fig. 6.—Patient with dementia praecox. Same as Figure 1. Two weeks' growth of beard; absence of hair from neck and sides of face.

SEX BEHAVIOR

An attempt has been made to approach the problem of sex in dementia praecox from the standpoint of behavior, somewhat according to the conception of behavior outlined by Watson.⁷ I have attempted, first, to determine what the actual sex behavior of persons developing dementia praecox has been. It is well recognized that in many cases the sex life has been abnormal, and that a sexual coloring is often

7. Watson, John B.: *Psychology from the Standpoint of a Behaviorist*, Philadelphia, J. B. Lippincott Co., 1919.

prominent in the clinical picture of the psychosis. However, no systematic observations seem to have been made to determine just what these persons have actually done in the way of sex behavior, how far they have advanced in the evolution of their sex life, whether or not an adult and socialized level of sex behavior has been reached, and whether this behavior at the adult level has been maintained.

Marriage in Dementia Praecox.—While marriage cannot be taken as an absolute criterion of the complete evolution of behavior in the realm of sex, yet the low marriage rate in males developing dementia praecox is rather striking. Pollock and Furbush⁸ report 2,232 male first admissions with dementia praecox in twelve states in 1919. Of these, 73.9 per cent. were single. From Table 2 it will be seen that the marriage rate for males developing dementia praecox is definitely below that of the general population, and that the difference is greatest in

TABLE 2.—MARRIAGE IN RELATION TO AGE

Dementia Praecox				General Population *	
Age on First Admission	No. of Cases ^a	Number Married	Married,† Percentage	Age	Married,† Percentage
15-19.....	76	1	1.3	15-19	0.6
20-24.....	108	1	0.9	20-24	20.9
25-29.....	52	6	11.5	25-34	63.3
30-34.....	57	16	28.0		
35-39.....	34	14	41.1	35-44	82.7
40-44.....	16	11	68.7		
Total.....	343	49	14.2		

* Middle Atlantic Division of United States, 1910.

† Widowed and divorced included.

the younger age groups. The dementia praecox patients referred to in Table 2 are those in whom physical sex development was studied.

Relations with the Opposite Sex.—More definite and concrete evidence of the degree of maturity attained in sexual life would be that of adult relations with the opposite sex. This question has been investigated in 137 unselected cases. The results are shown in Table 3. When the patient was mute, refused to answer, or if the answers were indefinite, evasive, or not considered dependable, the case was placed in the "not determined" group. In those indicated as "inadequate" one or more attempts at adult relations had been made with more or less success, but this level of behavior had not been established and maintained, for which various reasons were given. In those indicated as "adequate for a time," adult relations had been established and kept up for varying periods. In many of these, adult relations had not been indulged in for some months or years prior to first admission. The

8. Pollock and Furbush: Mental Diseases in Twelve States, 1919, Ment. Hyg. 5:353, 1921.

relation of this failure to the onset of psychotic symptoms is being made the subject of further observations.

Table 3 shows that 120 patients who were first admitted between 15 and 34 years of age answered the inquiry in a satisfactory manner. Of these 120 patients, 64.1 per cent. had never had sexual relations with the opposite sex. In an additional 15 per cent. relations had been attempted and carried out one or more times but had not been satisfactory, or for some other reason this form of sexual activity had been abandoned. Fear seemed to condition the reaction abnormally in certain of these cases, with a resulting panic.

Only 20.5 per cent. had ever reached an adult level of behavior and maintained it for even a short period either single or married, assuming that relations were kept up in all those who married. More detailed inquiry in several cases of the 12.5 per cent. of single persons who had kept up relations showed that such relations had usually been on the lowest prostitute basis. No further interest in the partner was

TABLE 3.—SEX BEHAVIOR IN DEMENTIA PRAECOX

Age on First Admission	No. of Cases	Single					Married, Cases
		Activity Not Determined, Cases	No Relations with the Opposite Sex		Relations Inadequate, Cases	Relations Adequate for a Time, Cases	
			Cases	Percentage			
15-19.....	30	3	22	81.4	4	1	0
20-24.....	53	10	29	67.4	6	7	1
25-29.....	34	3	17	54.8	5	4	5
30-34.....	20	1	9	47.3	3	3	4
Total.....	137	17	77	64.1	15%	12.5%	8.3%

manifest, and masturbation was practiced in the intervals. Even common law relations were avoided or not kept up. Heterosexual life in any socialized way seemed just out of reach. In this sense the behavior could scarcely be called adequate.

Early onset of the psychosis is the first point for consideration in explanation of these findings. The cases have been tabulated according to the age at the time of first admission rather than the age of onset of the psychosis. By careful study of these cases it might be found that the actual onset had been sufficiently early to explain the lack of sexual activity. It will be observed from the tables that the earlier the age on admission the higher was the percentage of cases showing sexual inactivity. But even in patients first admitted between 25 and 34 years of age there had been no relations with the opposite sex in over 50 per cent. To explain these simply on the basis of early onset, it would be necessary for the onset to have occurred from five to ten years before the first admission of the patient. This would indicate that the onset occurred at puberty or soon after, for it can probably be safely assumed

that most males accomplish heterosexual relations soon after puberty and usually repeat such relations at more or less frequent intervals. It would, therefore, seem that the findings cannot be easily or simply explained on the basis of early onset of the psychosis.

It did not appear that genuine moral or religious self-restraint had an influence, except possibly in a few cases, and in only a few were any such reasons offered. The frequency of masturbation and anti-social and immoral behavior often displayed by these patients speaks against such an explanation. A rather marked interest in religion is frequently manifested by persons developing dementia praecox. In many such cases it seems rather apparent that such abnormal interest is a part of an inadequate personality, or a kind of compensation for a sense of inferiority.

Further studies of sex from the standpoint of behavior are being made with an attempt to estimate, even in a rough way, the sex equipment of the individual in the way of instincts and emotions and of physical sex growth, to follow the evolution of the sex behavior reactions with this endowment, and, to ascertain what conditioning factors may have played a part. A perfectly constructed biologic mechanism, with complete instinctive and emotional equipment, and capable of functioning at the normal adult level of efficiency, has not been assumed, for it seems quite possible that such biologic factors as growth, metabolism and the internal secretions may play a part in determining sex behavior, and other behavior as well.

DISCUSSION

The findings indicate a disturbance of sexual development and a failure of sexual maturity which is most marked in patients admitted to the hospital during the years of puberty and adolescence. Patients admitted at an early age give the impression of an unevenness of sexual growth, the development of the secondary sexual characters lagging behind that of the testicles. The association of well developed genitalia with deficient secondary sexual hair in patients well past the age of puberty is shown in the photographs. These patients seem to lack the finishing touches of complete physical maturity.

The findings cannot be easily interpreted in terms of internal secretions. Gonads, and especially interstitial cells, are essential to complete sex maturity, but they are not self-sustaining. Other internal secretions may stimulate or activate them. Of interest in this connection is the patient shown in Figure 3, who had the physical make-up of the eunuch associated with well developed testicles.

Further evidence of a failure of complete sexual maturity, especially in terms of functional integration of the organism, is the large number of dementia praecox patients who have failed to reach the adult level

of sex behavior. That this failure is due to biologic inadequacy limited to the sex mechanism probably should not be inferred. The total functional inadequacy of many of these patients is seen in the well recognized inadequacy of their behavior in other directions, and the sex mechanism does not generate its own power, but derives it from the common source and merely transforms it to its own use. Adequate functional activity of the pituitary, thyroid, and suprarenals seems to be necessary for sex growth and function.

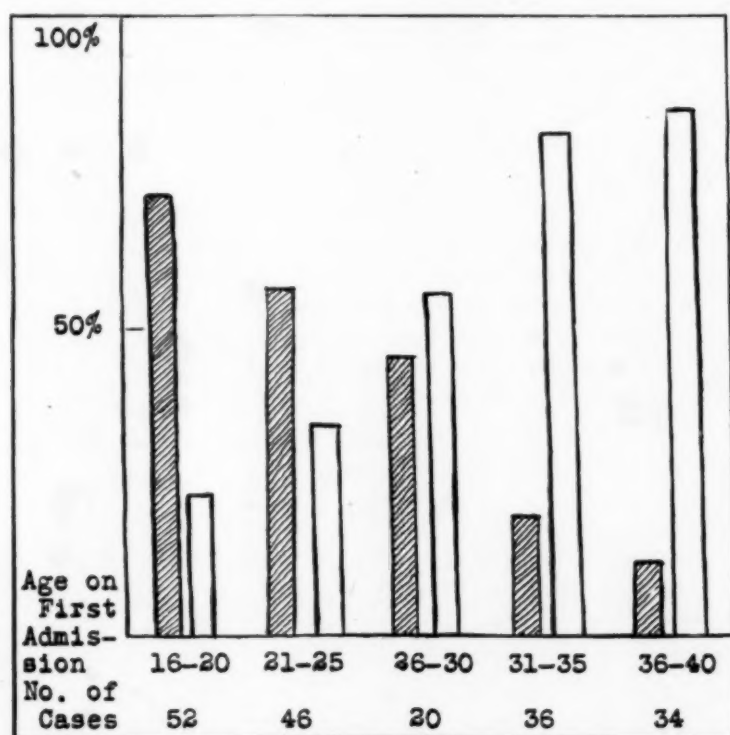


Fig. 7.—Diagnosis in relation to age. The dark areas represent catatonic and hebephrenic types, the light areas paranoid types.

The greater frequency of imperfect sexual maturity in young patients correlates well with the clinical types which are more frequent in early admissions. This relation in one series of patients included in this study is shown in Figure 7.

SUMMARY OF FINDINGS

1. The testicles of dementia praecox patients first admitted to the hospital between 16 and 20 years of age compared favorably in size with those of patients first admitted between the ages of 21 and 40.

2. Pubic hair of a definitely feminine distribution was present in 13 per cent. of patients first admitted between the ages of 16 and 20, and was still present in 13.4 per cent. after they were 21 years of age or over, but was present in only 2.6 per cent. of those first admitted between the ages of 21 and 40.

3. A definite deficiency of beard occurred more frequently in patients admitted early than in those admitted late. This deficiency persisted until after 21 in 34.6 per cent. of those first admitted between the ages of 16 and 20, and was still present in 21 per cent. of patients first admitted between the ages of 21 and 25 when those showing the deficiency had reached an average age of 25.8 years.

4. Deficient development of the secondary sexual hair did not depend on the size of the testicles, being associated with rather large testicles as frequently as with small ones.

5. The marriage rate of males developing dementia praecox was definitely below that of males in the general population.

6. Adult sexual relations with the opposite sex had never been accomplished by 64.1 per cent. of 120 dementia praecox patients who answered the questions in a satisfactory way.

7. Only 20.5 per cent. of the patients had reached an adult level of sex behavior and maintained it for even a short time, either married or single.

SPIROCHETE STAIN IN MULTIPLE SCLEROSIS

GEORGE S. STEVENSON, M.D.

Assistant in Neuropathology, Psychiatric Institute

WARD'S ISLAND, N. Y.

During the last ten years there has been an increasing interest in the etiology of multiple sclerosis. This has been stimulated by a growing body of facts which leads away from endogenous or idiopathic conceptions of this disease and gives increasing evidence of a living causative agent. Much has been written in reporting attempts to cultivate or demonstrate some organism by inoculation, and interest has centered about a spirochete as a possible cause of this disease. Animal inoculations have been made, and human tissue has been examined. A number of positive results have been obtained, but, in part these so disagree among themselves as to the characteristics of the suspected organism that different organisms must have been dealt with. Hauptmann¹ has given such a comprehensive review and criticism of the work reported previous to 1921 that further details will not be given here. Several reports have appeared since then. Birley and Dudgeon² have been unsuccessful in all attempts to isolate an organism or to transmit the disease to animals. They, however, feel that the evidence favors the inflammatory conception of the disease. Magnus³ has also been unsuccessful in animal inoculation experiments. Guy,⁴ who was the pioneer in experimental investigation of the disease, has done further work which makes him feel safe in concluding that multiple sclerosis is an infectious disease whose virus may sometimes be found in the cerebrospinal fluid. Schuster⁵ has recently announced the first demonstration of spirochetes in sections of human tissue. He used Jahnke's stain modified. His evidence in support of this claim is not convincing.

Since Jahnke⁶ through his modifications of previously existing methods of spirochete staining has so established the procedure as to make it dependable, it was considered worth while to undertake the study of a number of sections from cases of multiple sclerosis by his method.

1. Hauptmann: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **70**:300, 1921.

2. Birley, J. L., and Dudgeon, L. S.: *Brain* **64**:150, 1921.

3. Magnus, V.: *Etiology of Disseminated Sclerosis*, abst., *J. A. M. A.* **77**:2012 (Dec. 17) 1921.

4. Guy, W. E.: *Brain* **64**:213, 1921.

5. Schuster, J.: *Ztschr. f. d. ges. Neurol. u. Psychiat.* **73**:433, 1921.

6. Stevenson, G. S.: *Two Recent Improvements in the Staining of Spirochetes in Nervous Tissue*, *Arch. Neurol. & Psychiat.* **7**:349 (March) 1922.

Four cases were available for this study. All four had been shown by microscopic examination to possess the typical lesions of multiple sclerosis. In one case recently acquired by us only a few sections had been made. In all thirty-seven sections were examined; in Case 1 ten sections, Case 2 twelve sections, Case 3 eleven sections, Case 4 four sections. Sections were taken from many parts of the cortex, brain stem and spinal cord and one from the internal capsule. A few were taken with reference to an active process found by other stains. Some such process was present in twelve specimens examined. Most sections were taken at random and from widely scattered parts. These tissues were stained in four lots at different times. Three of these lots also included control tissue from parietic brains known to contain spirochetes. In each instance a good spirochete stain of this control tissue was obtained. In studying these tissues one section of each block was examined thoroughly and completely by the aid of a mechanical stage. High-dry magnification was mostly used. Whenever any suspicious object was seen it was examined more thoroughly with the oil immersion lens.

In all these sections no object was found which could be taken for a spirochete.

SUMMARY AND CONCLUSIONS

In the study of thirty-seven sections from four cases of multiple sclerosis stained by Jahnke's new method no spirochetes were found.

These studies indicate that spirochetes are absent or infrequent in multiple sclerosis tissue.

Spirochetes held to be associated with multiple sclerosis, if present, probably cannot be demonstrated by the most dependable silver stains now available.

Abstracts from Current Literature

THE DIAPHRAGM CENTER IN THE CEREBRAL CORTEX, AND HICCUP. KNAPP, Monatschr. f. Psychiat. u. Neurol. 50:333 (Dec.) 1921.

The ordinary movements of the diaphragm as they occur in respiration and phonation, beginning at birth are under the control of subcortical motor centers. Gradually, the cerebrum acquires a degree of control over these lower centers, as evidenced by the fact that the normal reflex automatic movement of the diaphragm can be to some extent voluntarily controlled; we can voluntarily increase or decrease our respiration, or for a brief period, even stop it altogether. Thus on physiologic grounds we are justified in assuming a cortical center for diaphragmatic movements. This center is intimately related functionally to the cortical centers for respiration and phonation; but they cannot be identical, as each can act independently of the others.

The diaphragm is a bilaterally acting muscle. It has been demonstrated for other muscle groups of this type (phonation, etc.) that there is bilateral cortical representation and bilateral control. Unilateral stimulation produces a bilateral effect, while unilateral destruction produces no effect, or a transient and incomplete paresis. So we may assume that the diaphragmatic center is a bilaterally represented and bilaterally acting center, each hemisphere having stronger influence over the contralateral muscle. One would therefore expect *a priori* an irritation of the diaphragmatic center in one hemisphere to produce a bilaterally symmetrical diaphragmatic contraction. Clinicopathologic observation shows that this is the rule. Exceptions to this rule occur (rarely) in both directions, irritation of one center producing contraction only of the contralateral half of the diaphragm, and destruction of one center producing contralateral paralysis.

Bulbar Hiccup: In the majority of cases, hiccup is independent of the cerebral cortex, and is dependent on direct or reflex stimulation of the centers in the medulla which control the automatic and reflex diaphragmatic movements. As an example of this type, we can consider hiccup resulting from an overloaded stomach. The dilated stomach presses on the sensory plexuses above and below the diaphragm, and sends sensory impulses up to the medullary centers and out by way of the phrenic. Sensory stimuli of this kind can come either by way of the vagus, or the sympathetic, nerve. In this general group belong the cases of hiccup due to disease of the stomach and bowel, to ileus and peritonitis, to disease of the uterus and adnexa, following kidney and prostate operation. Hiccup occurring in crises in tabes dorsalis may be due to direct irritation of the ninth and tenth nuclei in the medulla. Hiccup due to bulbar irritation may also occur in the course of cerebral diseases, such as tuberculous meningitis. Bruns mentions it in tumors of the medulla and tumors of the posterior cranial fossa. In all these groups of cases we have direct irritation of the medullary center.

Cortical Hiccup: In another group of cases, hiccup is a cortical focal symptom, due to the irritation of the diaphragmatic center in the cortex. The author reports four cases of his own and four from the literature. This type of hiccup is analogous to jacksonian epilepsy, and in four of the cases the diaphragmatic contraction alternated with contractions of other muscle groups of jacksonian type.

Prognosis: Formerly hiccup occurring in cerebral disease was considered a serious symptom. This is true of cerebral hiccup of bulbar origin, but is not so true of the cortical type.

Localization of Cortical Hiccup: Bergmark has reported a unilateral crossed paralysis of the diaphragm where the focus lay in and in front of the precentral gyrus. In his case the lesion extended from the top of the hemisphere to the upper part of the arm center. Therefore the diaphragm center must be between the leg and arm regions, where Sherrington puts the trunk zone, or in front of the leg and arm region, where most other investigators put the trunk centers. In some previous studies the author demonstrated that the arm and leg center touch, there being no room between them for the trunk centers. Therefore he believes the trunk and the diaphragm centers lie in front of the arm and leg centers where the centers for hip and shoulder meet. The weight of evidence from the whole group of cases is that the cortical centers for the diaphragm lie in the upper half of the foot of the second frontal convolution. According to an observation of Bechterew, the respiratory center lies very close to this same region.

Not all the cases corresponded to this localization. In one case the lesion involved the foot of the central convolution, and in two others the temporal lobe, especially the anterior portion. For this group the author does not assume a second center. He believes that in all this group, directly or by pressure, there is involvement of the foot of the precentral gyrus. Here lies the center for phonation, which is intimately associated with diaphragmatic movements, and which must be connected with the diaphragmatic center by extensive association tracts.

Thus we have two cortical zones from which a contraction of the diaphragm can be produced. The first lies in the foot of the second frontal convolution, in front of the lower part of the leg center and the center for shoulder movements, close to the center for the abdominal muscles and in the region of the inspiratory (respiratory) center.

A second center lies a considerable distance away. It is closely connected with the centers for phonation, chewing and swallowing, in the foot of the precentral gyrus, close also to the center for facial movements—all centers which have close relationship to the diaphragmatic and respiratory movements.

The first is undoubtedly the true cortical diaphragmatic center. The second is connected with this with numerous association fibers.

SELLING, Portland, Ore.

STUDIES IN EXPERIMENTAL TRAUMATIC SHOCK. V. THE CRITICAL LEVEL IN A FALLING BLOOD PRESSURE. W. B. CANNON and McKEEN CATTELL, *Arch. Surg.* 4:300 (March) 1922.

The authors begin this study by noting that in forty-five coincident determinations of blood pressure and carbon dioxid capacity, made on wounded men, the alkali reserve decreased with the falling arterial pressure. The respirations increased in rate as the alkali reserve diminished; they were, however, shallow and feeble, and in the authors' opinion were probably not effective in ventilating the lungs and thus lessening the concentration of carbon dioxid in the blood. The primary factor in the production of the low carbon dioxid capacity, the authors believe, is the marked fall of arterial pressure with a resulting diminution of the oxygen supply.

Experimental work on animals under conditions analogous to those of shock is discussed. A small slit was made in the pericardium of the animal and a cannula connected with a funnel by a rubber tube inserted; by means of this arrangement, physiologic salt solution was allowed to flow into the pericardial cavity. The output of the heart was directly controlled by the pressure of the fluid in the pericardium, and thus by raising or lowering the funnel any desired arterial pressure could be maintained. Artificial respiration and ether anesthesia were used. The alkali reserve of the blood plasma was determined each hour at various pressures.

The results obtained by this method showed that with a pressure reduced to 80 mm. of mercury, the alkali reserve was unaffected after one hour; at 70 mm. there was a slight reduction, averaging 3 per cent.; at 60 mm., there was a marked fall; at 50 mm. there was a still greater fall. The most rapid fall occurred during the first hour, after which it reached a stationary low level. The alkali reduction rapidly disappeared when the blood pressure was allowed to return to normal. Four animals bled to 20 per cent. of their calculated blood volume to produce a condition analogous to hemorrhage, with blood pressures reduced to 80 per cent., showed indications of inadequate circulation, which was less when the blood pressure was raised to 90 per cent. The authors conclude that the critical level, that is, the point beyond which oxidation is inadequate, is 80 mm.; accordingly, reduction of the alkali reserve is not likely to appear. These observations are corroborated by clinical evidence.

Morphin was found to modify the development of acidosis. When morphin was given before the pressure was lowered to 60 mm., the alkali reserve was not reduced; when it was given after the pressure was lowered to 60 mm. the alkali reserve, which had fallen, began to rise, and after two hours was largely restored. The authors suggest that morphin reduces the activity of tissues, lessens the demand for oxygen, and thus compensates for the smaller supply of oxygen in the sluggish blood flow.

When the blood pressure was reduced 60 mm., held for an hour, by intrapericardial compression, and then released, it rose to normal; when the procedure was repeated after an interval of an hour, the blood pressure did not rise so high, and after another hour it did not rise at all. This is interpreted to mean that injury to the vasomotor center has resulted, and after a prolonged low pressure, the maintenance of vascular tone has been impaired.

The authors review the previous work on the sensitiveness of various tissues due to a lack of oxygen. Muscle withstands anemia well; the brain is particularly sensitive. The pyramidal cells of the cortex are the most sensitive, chromatolysis being produced in eight minutes. The Purkinje cells of the cerebellum are next in sensitivity, while the medulla and sympathetic ganglions are more resistant. The work of E. L. Porter is cited, showing that the reflex arc disappears when the oxygen content of arterial blood is reduced to 4.5 per cent. by volume, and his conclusion that a lack of oxygen is more important than an accumulation of carbon dioxid in abolishing the capacity to transmit nerve impulses is noted. The authors quote these data with reference to their deduction that in the conditions of low blood pressure studied there is injury to the vasomotor center as a consequence of insufficient oxidation from a sluggish flow of blood.

The practical points following from the study are stated. The low blood pressure incident to shock must be combated at once. If the pressure falls below 80 mm., damage will result to the neurons, particularly in the vaso-

motor center; if the pressure is persistently low, the vasomotor center will be permanently damaged, and the pressure will remain low because of the lost capacity to maintain vascular tone.

VONDERAHE, Philadelphia.

CLINICAL STUDIES ON THE DEVELOPMENT OF SCHIZOPHRENIA.

I. ERWIN POPPER, *Monatschr. f. Psychiat. u. Neurol.* **50**:159 (Sept.) 1921.

The cause of schizophrenia is unknown, but certain factors contribute to its development which we can study. Considering the incidence of the disease, we find that beginning with the second decade there is a rapid increase in the number of cases, reaching a maximum at about the twenty-fifth year; after that it begins to fall, first slowly, then more rapidly. From the fortieth year on, the number of cases is small. If we separate the curves according to sex, we find that in the male the peak is a little higher, and the drop which follows the peak is sharper. After 40, it practically reaches the base line. In women the peak is not quite so high, and runs along fairly level from 25 to 35, dropping toward the base line more slowly. Practically all the cases occurring after the age of 40 are in women.

Hereditary Factors: A careful family history showed for 60 per cent. of the men and 61 per cent. of the women, cases of frank psychoses, marked anomalies of character, affectivity and general make-up "queer," psychopathic, neurotic, hysterical and alcoholic persons.

In studying the intellectual status of his schizophrenic patients, the author depends chiefly on the school record. The figures are as follows: Men: Intelligence good 36 per cent., medium 15 per cent., and poor 49 per cent. Women: Intelligence good or fair 66 per cent., poor 33 per cent.

Prepsychotic peculiarities of character, or affectivity, were shown by 43 per cent. of the men and 43 per cent. of the women. These figures are so high that they strengthen the view that preexisting affective anomalies are important factors in the development of schizophrenias.

Developmental anomalies on the somatic side occurred in 29 per cent. of the men and in 25 per cent. of the women.

Epilepsy and epileptiform symptoms of various kinds occurred in 15 per cent. of the men and 13 per cent. of the women.

Altogether, more than 60 per cent. of the patients showed hereditary factors, and 66 per cent. showed abnormalities of the prepsychotic make-up. Hereditary factors alone, without any abnormalities of prepsychotic development, occurred in 18 per cent. of the men and in 15 per cent. of the women. Prepsychotic abnormalities without any determinable hereditary factors occurred in 19 per cent. of men and in 17 per cent. of women.

In 15 per cent. of the men and in 22 per cent. of the women no hereditary factors and no prepsychotic abnormalities of character or disposition were found to determine the occurrence of the disease.

In considering the causation of schizophrenia, we have three possibilities: (1) that it is a purely exogenic disease; (2) that it is a purely endogenic disease; (3) that endogenic factors are essential, but that on these must be grafted certain exogenic causes.

Hereditary taint and abnormalities in the prepsychotic make-up are so frequent that we must recognize their importance in determining the development of schizophrenia. We realize that data of this type are more or less vague and ill defined. When we try to group them into a composite picture, we find that we cannot define a schizoid personality or a schizoid constitution. We

cannot select any one characteristic which would justify us in concluding that a person would become a schizophrenic. We cannot as yet recognize the disease in the making. And even when we accept these endogenic factors at their full value, we find that we must still attempt to explain the group of cases (one sixth of the total number) in which no such factors can be determined.

THE TRANSPLANTATION OF SKIN IN FROG TADPOLES, WITH SPECIAL REFERENCE TO THE ADJUSTMENT OF GRAFTS OVER THE EYES, AND TO THE LOCAL SPECIFICITY OF INTEGUMENT. WILLIAM H. COLE, J. Exper. Zool. **35**:353 (May 20) 1922.

Over the eyes of tadpoles of *Rana catesbeiana* and *R. clamitans* varying in length from 20 to 100 mm., opaque skin was transplanted to demonstrate the existence of any regulatory interaction between graft and skin tending to restore the function of the eye. In 66 per cent. of the operations in which tail skin was placed over the eye, the grafts were absorbed in a way which tended to expose the eyes. Back skin grafts were never so absorbed. Tail and back skin placed on other parts of the body were never absorbed. Tail skin grafts over hemispheres of glass or celloidin were absorbed in the same way. Back skin grafts so placed were not absorbed. Tail and back skin grafts over thin glass plates were not regularly absorbed. The foreign material is not the stimulus for absorption. The absorption over eyes and over the convex "artificial eyes" is caused by the curvature of the eyeball or the "artificial eyes." The more compact structure of back skin prevents its absorption. The curvature of the graft during healing causes a tension which is the mechanical stimulus for absorption. All tail skin grafts proliferated new tissue. The anterior and posterior ends of such grafts can regenerate a tail tip containing notochord and nerve cord. Following proliferation all grafts enter a state of equilibrium in which there is no increase in their size. There is no functional regulation of skin grafts over the eyes. This is the result of the high state of differentiation and specificity attained by the skin and the eye.

Integument from the tail, back or belly transplanted to another region of the same animal preserves its individual characteristics indefinitely. Homeo-transplants of skin preserve their individuality only temporarily, their tissues being replaced by regenerated tissue of the host. The integument of frog tadpoles is locally specific, and is self-differentiating when transplanted to new soil on the same animal.

Autotransplants of white belly skin on the back or tail acquire melanophores through the formation of pigment cells from epithelial cells in situ. Homeotransplants acquire melanophores through epidermal migration from surrounding skin, followed by formation of pigment in situ.

When the conjunctiva of frog tadpoles is extensively injured the regenerated tissue is pigmented, first by migrating epidermal melanophores and later by a mitotic multiplication of integumentary cells forming dermal melanophores and xantholeukophores. This is further proof that no correlation between the eye and the overlying integument exists in tadpoles 20 mm. or more in length.

Expansion of the dermal melanophores is caused by a low temperature, by darkness, by a 0.1 per cent. chlorotone solution, by anoxemia and by the low metabolic rate coincident with a moribund state. Contraction is caused by a high temperature, by light, and by a return of normal environmental condi-

tions after chloretone or anoxemia. Contraction is also coincident with recovery from a moribund state. Expansion or contraction usually requires about thirty-six hours for completion. It is probable that any stimulus lowering the metabolic rate of the whole animal causes expansion, and that stimuli causing an increase in metabolic rate cause contraction of the melanophores.

WYMAN, Boston.

ELEMENTARY CONVULSIONS IN CATATONIA. BAUSCH, *Monatschr. f. Psychiat. u. Neurol.* 50:319 (Dec.) 1921.

Through the work of H. Fischer, study of the convulsion has become a problem of its own, independent of the disease in which it occurs. The convulsion is not pathognomonic of any specific disease, but represents a type of reaction to various exogenic and endogenic traumas. This reaction type—the convulsive faculty—is not dependent on the brain alone but on a mechanism preformed in the entire body, corresponding to the mechanism which determines the general motor reaction forms of the organism.

The position which Vorkastner takes illustrates the general attitude toward the cases of convulsive seizures occurring in catatonia. Vorkastner asks the following questions:

1. Is there a dementia praecox with symptomatic convulsions?
2. Is there an epileptic psychosis simulating catatonia?
3. Is there a combination of two psychoses—catatonia and epilepsy?

The author considers that this way of looking at the problem is faulty and of itself interferes with the solution. It is due, in considerable measure, to the use of the term "epileptic seizure" or "epileptiform seizure" for the convulsion, thus substituting the idea of a disease entity for a mere symptom. If we accept Fischer's thought, separating the convulsive seizure from the convulsive disease, and using a name—elementary convulsion—independent of any disease concept, we come to a different formulation of the problem.

1. Are the elementary convulsions a specific symptom of dementia praecox, more particularly the catatonic type; or (2) are we dealing with purely symptomatic convulsions, such as occur in other endogenic diseases (diabetes, exophthalmic goiter, etc.)?

Looking at it in this way, Vorkastner's questions 2 and 3 are automatically eliminated, and we can dispense with the idea of any such combination disease as catatonia-epilepsy (Vorkastner) or "epileptic form of catatonia" (Urstein). The author illustrates his point of view with two case reports of convulsive seizures occurring in cases of dementia praecox.

In the first case there was a family history of epilepsy (paternal grandmother), as well as certain stigmas during childhood, such as bed wetting, abnormal fears, etc. The patient was solitary, unadaptable and shy. At 25 the psychosis began, and developed into a typical catatonia. During the course of the disease, elementary convulsions occurred. The author believes that they are the result of a constitutional component.

In Case 2 there is no history of convulsive diseases in the family. The patient had convulsions in the first months of life, but otherwise developed normally. Change in character which represented the onset of his psychosis began at puberty. It developed into a typical dementia praecox (catatonia), during the course of which a number of elementary convulsions occurred. For a time these dominated the picture, being classed sometimes as hysterical, sometimes as epileptic. The whole personality gave no suggestion of an

epileptic character. On the other hand, the emotional dulling was progressive during the period of observation. Bausch calls attention to a curious variability in the motor manifestations of the disease, stupor and akinesia being replaced at various times by excitement and elementary convulsions. The convulsions occurred only at the beginning or end of a stupor period. Often they started a period of general motor excitement, with impulsive acts, verbigeration and lively hallucinosis. This relationship between cataleptic phenomena and stuporous conditions on the one hand, and convulsions and motor excitement on the other, points to a relationship between these various manifestations of motility.

In both cases, then, there is a constitutional convulsive factor (heredity in one, convulsion in early life in the other). In both, the dementia praecox is complicated by convulsions, which the author considers as symptomatic convulsions in catatonia.

We avoid much difficulty if we consider the convulsion independently of any disease concept. We must look on the convulsion merely as one of the many types of motor disturbance occurring in catatonia, and consider this type of case as a peculiar constitutional coloring of the catatonia. As we have symptomatic convulsions in other diseases, so we have them here, and we are not justified in assuming a combination of two diseases.

SELLING, Portland, Ore.

STUDIES ON THE VISCERAL SENSORY NERVOUS SYSTEM. XIV.
THE REFLEX CONTROL OF THE CARDIA AND LOWER
ESOPHAGUS IN MAMMALS. A. J. CARLSON, T. E. BOYD and J. F.
PEARCY, Arch. Int. Med. 30:409 (Oct.) 1922.

The authors point out that a knowledge of the reflex control of the cardia and lower esophagus is important in explaining spasms of the cardia and esophagus in man. The animals used in the experiments were cats and dogs. One dog was provided with a permanent gastrostomy and an esophageal fistula in the neck, and was used for the study of reflexes to the cardia from the mouth, pharynx and stomach, the action of cocaine on the cardia, etc., without anesthesia. In the acute experiments, the animals were anesthetized with ether. Gastrostomy and fistula of the esophagus were made for fixing a cardiometer in place, and usually a balloon was also placed in the stomach for recording the gastric tonus. The procedure after this was as follows: (1) conduction of experiments under constant, but light ether anesthesia; (2) ether anesthesia continued with curare and artificial respiration, for elimination of skeletal and respiratory reflexes; (3) animal decerebrated and ether anesthesia discontinued; (4) the foregoing conditions followed with chest and respiratory mechanism intact, or with chest opened for artificial respiration, the phrenic nerve sectioned and the diaphragm dissected away from the esophagus to eliminate possible errors from spasm of the diaphragm.

In normal dogs, not under anesthesia, the tonus of the cardia was temporarily inhibited by stimulation of sensory nerves in the mouth and pharynx and by stimulation of nerves in the gastric mucosa; it was increased by sudden distention of the walls of the stomach, by intravenous injections of small quantities of cocaine, by some factor other than the acidity of the gastric contents during gastric digestion. The tonus of the cardia was found to run parallel with the tonus and hunger contractions of the empty stomach.

In dogs and cats under light ether anesthesia, ether and curare or decerebration, it was found that reflex inhibition or contraction of the cardia and

lower esophagus can be initiated by the stimulation of any sensory nerve skeletal or visceral; that when the vagi are intact, stimulation of the sensory nerves in the mouth, pharynx, esophageal and gastric mucosa induce on the whole inhibition of the cardia, followed by contraction; that stimulation of the afferents from the abdominal viscera (gallbladder, intestine, urinary bladder, central end of one of the splanchnic nerves) induces, on the whole, reflex contraction of the cardia and lower part of the esophagus, even when both vagi nerves are sectioned; that when the tonus of the cardia is feeble, the motor reflexes into the cardia predominate, and when the cardia is hypertonic, the inhibitory reflexes prevail; that strong stimulation of the abdominal viscera of the central end of the splanchnic nerve may cause strong spasm of the cardia and lower esophagus lasting from ten to thirty minutes.

The authors note the fact that the tonus of the cardia being inhibited reflexly by chemical and mechanical stimulation of the sensory nerves in the mouth may have some practical application. Bitters or acids in the mouth may reflexly help to inhibit the milder forms of reflex cardiospasm.

VONDERAHE, Philadelphia.

LOCALIZATION OF APRAXIA. FORSTER, *Monatschr. f. Psychiat. u. Neurol.* 50:1 (July) 1921.

In 1912, the author reported a case of bilateral apraxia with associated symptoms pointing to the left-hand center (right hemiparesis, jacksonian attacks beginning in the right hand). Operation revealed a glioma lying just beneath the surface at the base of the second frontal convolution. The findings were confirmed by postmortem examination. In presenting the case at the time, the author assumed that the apractic symptoms were referable to the frontal lobe lesion. The possibility of their being the result of a lesion of the precentral gyrus could not be excluded.

Subsequent study of the case with serial sections demonstrated that there were three tumors: one, already described, involved the foot of the second frontal convolution; a second lay in the substance of the first frontal convolution, a little posterior to this; the third involved the anterior portion of the corpus callosum.

In his original presentation of the case, the author said he believed that it proved frontal lobe lesions capable of causing apraxia. His subsequent studies failed to bear out this contention. Areas of degeneration were found in the central convolution so that certain connections between frontal lobe and central convolution must have been lost. Furthermore, the central convolutions were cut off from the corresponding areas of the right hemisphere by the callosal tumor. There was no obvious disturbance in the connections between the central convolutions on the one hand, and the occipital and temporal convolutions on the other. But the existence of areas of rarefaction in the subcortical zone of the central convolutions compels the admission that such breaks may have existed.

Kleist, as a result of the study of war material, believes that limb-kinetic apraxia results from involvement of the precentral gyrus, whereas ideokinetic apraxia results from left-sided parietal lobe lesions. Left-sided apraxia in right-sided paralysis occurs (with one exception) only when the paralysis is accompanied by sensory disturbances and touch agnosia. Kleist concludes that the impulses regulating the functioning of the left arm reach it, not through

the left precentral gyrus, but from the left parietal zone. If the parietal zone is undamaged, there must be involvement of the callosal fibers coming from the depths of the parietal lobe, or of the corpus callosum itself.

In the author's case, neither parietal lobe nor deep callosal fibers, nor the posterior portion of the corpus callosum were involved. But the tumor of the anterior portion of the corpus callosum may have thrown the posterior portion out of function by pressure.

The bilateral apraxia in this case may be explained as due to lesion of the left precentral gyrus; and the left-sided apraxia is explainable in addition on the basis of damage to the corpus callosum in this region. The assumption that the parietal lobe either through pressure or through injury to its callosal fibers may have in part been responsible for the loss of function cannot be entirely excluded.

SELLING, Portland, Ore.

THE PALM-CHIN REFLEX. A. RADOVICI, *Ann. de méd.* 12:56 (July) 1922.

The author points out that the cutaneous reflectivity of the upper extremity is less than that of the inferior extremity. This difference finds its explanation in the fact that movements of the upper extremity are eminently voluntary, since the arm serves to bring about movements of precision toward a determined end. In contrast, maintenance of the erect posture and the automatism of walking are accomplishments pertaining to the lower extremity, and carry with them the greater development of reflexogenic phenomena. The skin of the thorax, arms, forearms and palmar surface of the hand are classically considered as exempt from cutaneous reflexes. However, two years ago, in collaboration with Marinesco, the author described the existence of a palmar cutaneous reflex. The excitation of the palm of the hand produces a contraction of the muscles of the chin on the same side. This so-called palm-chin reflex exists in about 60 per cent. of normal persons. Normally, there is often a difference between the right and left reflex in favor of the left. It is suggested that the right arm is more strongly differentiated from the view of voluntary motility and therefore presents less reflex activity than the left.

The palm-chin reflex is produced by a rapid stroking of the thenar eminence with a sharp point, and consists of a quick contraction of the muscles of the chin on the same side, producing a marked lifting of the lower lip. There must be preliminary relaxation of the lip and the mouth must be half open.

The examination of the palm-chin reflex gives indications for the diagnosis of the site of lesions of the pyramidal tracts in their superior portions. The unilateral exaggeration of the reflex indicates a bilateral supranuclear lesion of a pyramid above the seventh nerve nucleus. A bilateral exaggeration of the reflexes indicates a bilateral supranuclear lesion of the pyramids: pseudobulbar paralysis, amyotrophic lateral sclerosis and Little's disease. A nuclear or peripheral lesion of the facial nerve abolishes the reflex on the side of the lesion. A high cervical spinal lesion above the brachial swelling can bring about a modification of the reflex. In one case the palm-chin reflex was not present, but excitation with a needle of the palm or thenar eminence produced an extension of the thumb (analogous to the Babinski sign).

A total lesion at the brachial swelling abolishes the reflex, while a partial lesion, which does not affect the eighth cervical and first dorsal segments can

produce a modification of the reflex, so that palmar excitation causes muscular contraction of the pectorals, supplied by segments which are unaffected.

The existence of the reflex in high dorsal lesions proves that the lesion is inferior to the first dorsal, the lowest brachial segment.

DAVIS, New York.

RESULTS OF PUNCTURE OF THE CORPUS CALLOSUM. POHLISCH, *Monatschr. f. Psychiat. u. Neurol.* **50**:251 (Nov.) 1921.

The author gives a summary of the results of callosal puncture, introduced by Anton and von Bramann, in 1908. The operation was recommended by its authors as a relatively simple method of procuring permanent ventricular drainage, applicable as a palliative measure to practically all conditions of intracranial pressure increase. Pohlisch reports ten cases of his own, and has reviewed 550 cases from the literature. He compares the method and its results with ventricular puncture and decompression. Spinal puncture is not considered because of the dangers attendant on its use in case of pressure increase.

Operative Procedure: Callosal puncture is simpler than decompression, but no simpler than ventricular puncture. There are definite possibilities of trouble in the form of hemorrhage, thrombosis and collapse resulting from too rapid pressure reduction. In view of these complications, the author considers the procedure hardly less serious than ventricular puncture or decompression.

Diagnostic Significance: Occasionally one can palpate with the cannula a tumor lying in the wall of the ventricular cavity. Aside from this, callosal puncture offers no advantages over ventricular puncture or decompression.

Therapeutic Results: (a) Hydrocephalus. Perhaps one third of all cases are favorably influenced. Focal symptoms show little improvement, but the general pressure symptoms (headache, dulness, vomiting, vertigo, convulsions), and especially the choked disk, may be considerably improved. In a number of cases it was possible to save vision. In a few cases more permanent results were obtained. The question of permanency of results is bound up with the question as to whether the drainage canal through the corpus callosum remains open. A small number of cases have been studied at necropsy. In one, the puncture was found closed after seven days; in another, it was open after two and one-half years. The author's conclusion is that in hydrocephalus, callosal puncture has no advantage over single or repeated ventricular puncture. (b) Brain tumors. In this group, as in the preceding, callosal puncture has no advantage over ventricular puncture. (c) Epilepsy, tumor skull, migraine. Anton and von Bramann suggested the use of callosal puncture in these groups of cases, but there is nothing to indicate that it is of any value. (d) Serous meningitis. According to Paye, callosal puncture has been of value in the treatment of cases of traumatic serous meningitis. His records did not indicate whether the benefit was due to the immediate pressure relief or to permanent drainage. Others have obtained similar results in the same type of case by ventricular puncture, and even by spinal puncture.

SELLING, Portland, Ore.

THE BASAL METABOLISM IN NONTOXIC GOITER AND IN BORDERLINE THYROID CASES, WITH PARTICULAR REFERENCE TO ITS BEARING IN DIFFERENTIAL DIAGNOSIS. JAMES H. MEANS and HARRIET BURGESS, *Arch. Int. Med.* **30**:507 (Oct.) 1922.

The authors analyze the data presented by basal metabolism determinations on 1,000 patients, with reference to the diagnosis of thyroid activity.

Five groups of cases are discussed in this paper. Cases showing subnormal metabolism, lower than minus 10 per cent., represent the first group. Low basal metabolism occurred in all untreated patients, numbering twenty-six, with myxedema and cretinism. Sixty-three others presented a low metabolic rate; 84 per cent. of these either had endocrine disorders or presented some clinical suggestion of endocrine disease; in the remaining, the subnormality in rate was slight. The endocrine diseases other than thyroid included suprarenal and pituitary abnormalities, and a probable case of Addison's disease. In anemia, polycythemia and leukemia, subnormal metabolism was found to be a distinct rarity. In the second group are placed cases with a supernormal metabolism. Of 300 cases showing clinical signs and symptoms of hyperthyroidism, only one failed to show a metabolic rate of plus 10 per cent.; this patient had mild symptoms. Supernormal metabolism was found in blood diseases, in leukemia and severe anemia and in 24.4 per cent. of endocrine disorders other than hyperthyroidism. Twenty cases, nonendocrine in character, presented increases in metabolism; these included a woman seven months pregnant, patients with malignant lymphoma of the neck, osteitis deformans, paroxysmal tachycardia and probable chlorosis. In a case of paralysis agitans, the first determination was plus 23 per cent.; the second determination made a week later when the patient was more quiet was minus 2 per cent. In the third group are 102 cases of nontoxic goiter. All of these cases gave metabolic rates within normal limits. A fourth group is made up of seventy patients suspected of having hypothyroidism, most of whom had normal metabolic rates; those that had subnormal rates improved under the administration of thyroid extract. A fifth group was made up of 290 patients suspected of having hyperthyroidism; two thirds of these gave metabolism rates with normal range, one third were supernormal. Errors, the authors note, are always in the direction of higher readings, so that a normal rate in the presence of a suspected hyperthyroidism rules out this condition.

On the basis of the evidence presented the authors conclude that, provided certain well recognized causes for increased metabolism, such as fever, acromegaly, leukemia and severe anemia, are excluded, the finding of an increased metabolic rate is strong presumptive evidence of hyperthyroidism; and that, provided causes for decreased metabolism, such as starvation, hypopituitarism and hyposuprarenalism, are excluded, a low metabolic rate is strong presumptive evidence of hypothyroidism.

VONDERAHE, Philadelphia.

TETANY. A REPORT OF CASES WITH ACID-BASE DISTURBANCE.
SAMUEL B. GRANT, Arch. Int. Med. **30**:355 (Sept.) 1922.

The cases of tetany reported occurred in adults. In all instances studied there was a marked increase in the sodium bicarbonate of the blood plasma. Three of the cases occurred in obstruction of the stomach; in one of these cases the blood chlorid was markedly decreased, but the blood calcium was within normal limits. Two cases occurred following overdosage with sodium bicarbonate. In one of these cases the soda was given in the course of a Sippy treatment for gastric ulcer. On the fifth day a prickling sensation of the hands developed, followed later by a flexion spasm of the fingers; when alkali was discontinued no further spasm occurred. The plasma carbon dioxide capacity the following morning was 90.5 volume per cent., and at this time there was a positive Trousseau sign, flexion spasm of the fingers, adduction of the thumb and a faint Chvostek sign. When the treatment was continued

with smaller doses of alkali bringing the plasma carbon dioxid capacity to 71 volume per cent., the signs of tetany disappeared. In the other case sodium bicarbonate was given intravenously and by Murphy drip in a case of severe acidosis accompanying toxemia of pregnancy. A rise of the carbon dioxid capacity from 19.5 volume per cent. to 27 volume per cent. appeared. Sodium bicarbonate, 2 gm. every two hours, was given by mouth in addition. Three days after admission the carbon dioxid capacity was 67.5 per cent., and the following day the patient developed carpal spasm with twitching about the mouth. At this time the carbon dioxid capacity had risen to 87 volume per cent.

The last case reported accompanied hysteria in a nurse, who, failing in her examinations, developed an attack characterized by deep and rapid breathing, after which she complained of numbness and tingling in her fingers. After stopping the hyperpnea, the sensations disappeared. Later the rapid, deep breathing returned, followed by tingling and numbness and later by flexion spasm of both hands. When she was persuaded to breathe quietly, the spasm subsided. Trousseau's sign was positive. Unfortunately, the carbon dioxid capacity of the plasma was not determined; the explanation suggested is that the hyperpnea "washed out" the carbon dioxid from the blood, leaving a relative excess of sodium bicarbonate.

VONDERAHE, Philadelphia.

CONTRIBUTION TO OUR KNOWLEDGE OF LESIONS OF THE
SUBTHALAMIC REGION. BRESOWSKY, *Monatschr. f. Psychiat. u.*
Neurol. 50:302 (Nov.) 1921.

A patient, aged 39, had an attack of influenza followed by a right hemiplegia and mental symptoms. Neurologic examination, except for the usual findings in hemiplegia, was negative. One month later left-sided ptosis developed, eye muscle involvement and hemianopsia, all transient in character. Ten days later there was definite protrusion of the right eyeball. This unilateral exophthalmos gradually increased in degree. There were repeated convulsions. Death came suddenly, two and a half months after admission.

Necropsy revealed an encapsulated abscess of the left thalamus, which extended downward on the same side, destroying the red nucleus and part of the substantia nigra, and rupturing caudal to the corpus subthalamicus. Practically the whole thalamus, excepting the ventro-caudal and ventro-medial portion in the neighborhood of the corpus subthalamicus, was destroyed. The corpus subthalamicus itself was not involved.

The chief interest in the case centers in the author's interpretation of the right-sided exophthalmos. He discards the idea that it might be a distant symptom. He looks on it as a strictly focal symptom, due to irritation of the subthalamic area. It developed one month before exitus, at a time when, according to the history, the abscess was sinking from the thalamus toward the subthalamic region. This was indicated by the development of midbrain symptoms.

The author points out that for a long time the central gray of the mid-brain has been associated with the central origin of the sympathetic system. Karplus and Kreidl demonstrated in experimental animals that stimulation of a certain point in the subthalamic region would produce the same symptoms as stimulation of the cervical sympathetic: dilation of the pupil, widening of the lid slit, and exophthalmos. In their experiments, the symptoms were chiefly contralateral. In the author's case, the exophthalmos was also contra-

lateral. The same was true of a similar case reported by Schrottenbach. Gerstman has reported one with homolateral symptoms. According to the author's case, the crossing of the fibers must be caudal to the subthalamic region, not oral as Gerstman believed.

SELLING, Portland, Ore.

A PRELIMINARY STUDY OF THE PRECIPITATING SITUATION IN TWO HUNDRED CASES OF MENTAL DISEASE. EDWARD A. STRECKER, *Am. J. Psychiat.* **1**:503 (April) 1922.

This is a study of a fundamental psychiatric problem, that of the "precipitating situation," and incidentally of interest to those who in compensation, military and legal cases have to give reasons for their belief that a given incident is or is not a cause of a following psychosis.

In 100 cases each of dementia praecox and manic-depressive psychoses, Strecker gives in parallel columns a condensation of the psychic and physical situation at the onset of the disease and an opinion as to the importance of the special situation as a precipitant of the attack. Of 200 patients, 107 combined psychic and organic factors, forty apparently followed somatic disturbances alone, thirty-nine followed psychic disturbances, and fourteen failed to reveal any reason for the breakdown.

Apparently fully adequate precursors of breakdown appeared in thirty-two manic-depressive and in twenty dementia praecox patients, while no stress appeared before the onset in only two manic-depressive and in twelve dementia praecox patients. In both illnesses there was some evidence that the emotional tone proper to the important prepsychotic situation was carried over into the psychosis itself. It was suggested also that the accumulation of mental and physical strain over a considerable period of time may exceed in importance a single dramatic event. Mental diseases in the family and abnormal personalities appeared most frequently when the precipitating situation seemed trivial. In manic-depressive cases recurrences were also found most often when the original unfavorable elements in the situation were doubtful, insignificant or absent.

All careful clinical work in this direction, and even careful speculation, is valuable. We have come to a time when the study of physical aspects of a psychosis need not turn us away from its mental aspects. In the situations in this paper, how can we be sure that "loss of weight" is not a different side of "an unhappy marriage," or separate the worries about illegitimate pregnancy from the exhaustion of childbirth? So that the crux of Dr. Strecker's paper is not the question of mental against physical, but the question, "Of what importance was any particular set of factors in the onset?"

BOND, Philadelphia.

EXPERIMENTAL DIABETES INSIPIDUS. PERCIVAL BAILEY and FRÉDÉRIC BREMER, *Arch. Int. Med.* **28**:773 (Dec.) 1921.

This work describes systematic punctures of the hypothalamus in dogs, with observations on the symptoms following the operations. In operative study of the hypophysis this area is liable to injury, so that a study of its function becomes important in interpreting the results of previous experimentation.

The hypothalamus was explored by means of the lateral route; in each instance the hypophysis was left intact. Puncture of the parainfundibular

region of the hypothalamus in every one of the thirteen dogs produced polyuria which appeared within the first two days following the operation; in extensive lesions the polyuria became permanent.

The polyuria induced in the dogs was such that concentration of urine occurred when fluid intake was restricted or when the pituitary extract was injected subcutaneously or in the presence of fever, while excessive polyuric action was noted after the administration of chloride; the theobromin effect was absent. These reactions, the authors note, are found in diabetes insipidus of man. Thirst, with an increased intake, often appeared before the polyuria, but was not constant; in comatose animals polyuria appeared without intake of water. In animals whose kidneys had been denervated the polyuria persisted without essential change. In two cases in which the tuber cinereum was injured, a cachexia "hypophyseopriva" with genital atrophy developed; in two other similar cases with injury to the tuber cinereum there was an insidious development of adiposogenital dystrophy; in every instance there was a persistent polyuria. Animals with an extensive lesion of the tuber cinereum either died in a short time or after a period of coma and convulsions. Glycosuria was inconstant in its appearance.

The authors perceive clearly that this work questions the reliability of previous studies leading to the widely accepted opinions that adiposogenital dystrophy is due to a deficiency in the secretion of the anterior lobe of the pituitary and polyuria to a deficiency of the posterior lobe secretion. For this previous teaching they substitute the tuber cinereum as being concerned with the syndrome of adiposogenital dystrophy and the hypothalamus with polyuria. In the discussion of their observations they defend this view with considerable vigor and answer the objections which may be raised against it.

VONDERAHE, Philadelphia.

HEMIPLEGIA WITH HEMICHOREA. A. REMOND and H. COLOMBIES, *Rev. de med.* **39**:107 (Feb.) 1922.

This is a clinical and necropsy report of a case showing combined hemiplegia and hemichorea. A woman, aged 70, was suddenly aware of an intense tingling of the entire left side of her body; she then rapidly lapsed into unconsciousness and became hemiplegic (left-sided). At the moment that consciousness was lost, irregular disordered movements of moderate intensity began in the left arm. The face was animated by continuous spontaneous movements predominating on the left. Both the coma and the choreiform movements remained constantly until death after three days. The left arm, flaccid at the shoulder, showed a marked tonic of the forearm. The left leg, though flaccid, showed a positive Babinski sign. This sign was not present on the right. The tendon reflexes were not obtained on either side. The extremities of the right side presented normal tonus. The pupils were myopic and gave no reaction to light. The spinal fluid was transparent, not under pressure, and was not abnormal on examination.

Necropsy revealed a cerebral hemorrhage "the size of a large nut," situated just under the cortex in the superior portion of the perirhinal area. It was sharply limited, did not inundate the ventricles and did not attain any of the gray nuclei. Additional sectioning of the brain did not reveal any other lesions either recent or cicatrized.

The authors point out that such a hemiplegic and hemichoreic association is rare, and that incertitude holds regarding the lesion capable of producing it. They consider, however, that their case correlates with the ideas of Pick

and Kahler, who incriminate "an irritation of pyramidal tract fibers or of motor centers." The perirolandic cortex, caught between the cranial vault and the hemorrhage mass, received a constant irritation, made evident by motor reactions. They are carried only by the fibers not sectioned by the lesion, in their case, the fibers supplying the face and arm.

Touche has written that in almost all cases of hemiplegia with hemichorea there are multiple lesions. But the case reported had only one lesion, sharply delimited.

DAVIS, New York.

EXPERIMENTS ON THE DEVELOPMENT OF THE CRANIAL
GANGLIA AND THE LATERAL LINE SENSE ORGANS IN
AMBLYSTOMA PUNCTATUM. L. S. STONE, J. Exper. Zool. **35**:421,
(May 20) 1922.

The experiments were for the purpose of extending knowledge concerning the part which placodes and neural crest play in the formation of cranial ganglions and nerves and also to determine the extent of their contribution to the formation of the mesoderm and the exact origin and fate of the "mesectoderm" tissue. Above the optic vesicle in early stages there is an elongated ophthalmic placode which gives off cells to the formation of the ophthalmic ganglion. Near the anteroventral border of the supra-orbital lateral line primordium is a small gasserian placode of brief duration. Close to the anterior border of the auditory placode is a prominent placode elongated toward the dorsal extremity of the hyomandibular cleft. Removal of this gives rise to the absence of the supra-orbital line of sense organs and a large part of the seventh lateral-line ganglion. The supra-orbital primordium of lateral-line sense organs arises from the anterior extremity of the seventh lateral-line ganglion placode. The supra-orbital, infra-orbital and hyomandibular primordia of lateral-line sense organs have separate seats of origin. The ventral hyomandibular and mandibular groups of lateral-line organs appear to have separate seats of origin. The epibranchial placodes of the seventh, ninth and tenth segment give off cells which become incorporated in the visceral ganglions. Removal of ectoderm in the region of the ninth and tenth segments results in a complete absence of lateral-line ganglions. Cutaneous as well as visceral sensory fibers are absent. The neural crest cells, from the dorsal portion of the neural tube, can be distinguished as early as the closure of the neural folds. The wandering mass of "mesectoderm" is of neural crest origin and is not augmented by a contribution from cells of the lateral ectoderm. The hyohyals, ceratohyals, ceratobranchials, epibranchials and first basibranchial are formed from the wandering neural crest in the branchial and hyoid regions. In the trigeminal region the neural crest cells form the quadrate and mandibular cartilages. The neural crest in the trigeminal region which migrates over the anterior border of the optic vesicles apparently gives rise to the anterior portion of the trabeculae.

WYMAN, Boston.

CLINICAL STUDIES ON THE CAUSE OF SCHIZOPHRENIA. II.
ERWIN POPPER, Monatschr. f. Psychiat. u. Neurol. **50**:235 (Oct.) 1921.

In the foregoing paper the author discussed the occurrence and significance of endogenic factors in the development of schizophrenias. He points out that endogenic factors, as represented by heredity, disposition and prepsychotic personality, are more or less vague in character and difficult to evaluate.

Nevertheless, the frequency with which they appear in the histories of schizophrenias is such that we must recognize their importance in the development of the disease. In one sixth of all cases in this series, no endogenic factors could be determined, and it is this group particularly that forces us to a consideration of exogenic factors. Here, again, we encounter difficulties. A mere time relationship between a trauma or shock and the development of the disease is not sufficient; we must demonstrate that some more intimate connection exists. A further difficulty lies in the fact that not every psychosis which resembles schizophrenia actually belongs to that disease group. It is probable that some of the cases in which exogenic factors play an especially prominent rôle are not true schizophrenias, but reactive psychoses of different types. Summarizing his studies, the author concludes that we must assign to endogenic factors a primary significance in the development of schizophrenias. But considering the fact that one sixth of all cases show no endogenic factors, together with the frequency and intimacy with which exogenic injuries of one kind or another are associated with the outbreak of the disease or one of its episodes, we must admit that exogenic factors do have an etiologic significance.

SELLING, Portland, Ore.

TUBEROSE SCLEROSIS AND HYDROCEPHALUS AND THEIR ASSOCIATION WITH PRECOCIOUS PUBERTY. K. H. KRABBE, *L'Encephale* 17: No. 5, pp. 281-289; No. 7, pp. 437-445; No. 8, pp. 496-506.

The etiology and pathology of premature sexual development are not clearly understood. In this paper, Krabbe discusses precocious puberty and its association with cerebral affections and, in particular, certain forms of hydrocephalus, tuberoses sclerosis and pineal tumors. Precocious puberty due to tumors of ovarian or testicular origin forms a group of cases not germane to the author's study.

Numerous cases of several types are presented in detail, and there is a brief treatment of the literature bearing on these cases.

Krabbe points out that the presence of precocious puberty in certain conditions is no proof that it is caused by them. In cases of pineal tumor, necropsies have too frequently been limited to the head. In Krabbe's experience with pineal tumors, pathologic changes may be found elsewhere in the body, particularly in the endocrines. Moreover, in pineal tumors other things than the destruction of the pineal gland must be considered as possible factors in the production of precocious puberty—secretion of particular substances of the tumor involving the gland, action à distance on the pituitary and action on the cerebral sympathetic centers about the third ventricle.

Both from his own cases and from the literature Krabbe concludes that these local brain diseases are evidence of a general malfunction or disorganization of certain groups of tissues and organs and that precocious puberty is also evidence of this same anomaly. However, it may depend not only on the underlying anomaly, but on the presence of one or more other associated factors, such as pineal destruction or disturbed function in the cerebral portion of the sympathetic system. Krabbe emphasizes that any of the three of the cerebral conditions with which his paper deals may be present without distinct premature sexual development. This is particularly true in cases of pineal tumor and hydrocephalus. In fact, he has observed that hydrocephalus, as well as tumor, or destruction of the pineal gland, may coexist without consequent precocious puberty.

HYSLOP, New York.

TRANSITORY AGNOSIA OF AN INFANT IN REGARD TO ITS ARMS.

AUGUSTE TOURNAY, *Rev. neurol.* **29**:580 (May 4) 1922.

This report gives detailed daily observations on the lack of recognition of its hands (agnosia) by an infant from the age of 115 days to that of 141 days, as well as daily observations on the disappearance of the Babinski sign of infancy. The relation of these observations to the anosognosia of certain persons with left hemiplegia, described in 1914 and 1918 by Babinski, is discussed.

It was noticed that the child at the one hundred and fifteenth day began to take definite notice of its right hand, while it paid absolutely no attention to its left. The child was and is quite normal in every way, and is now 2 years and 4 months old. Vision in all fields was and is normal. This agnosia, involving the left hand and not the right, continued for about twenty-six days, after which the child began to become interested in and to take notice of its left hand. From that time on it was equally interested in both. Therefore for 115 days of its life it had no apparent power to appreciate itself (hands) or to differentiate its hands from the outside world. At this time it became conscious of its right hand as belonging to itself. After a period of twenty-six days, it became conscious of its left as belonging to itself.

One hundred and eighty-one days after birth, stimulation of the feet for the first time showed flexion of the great toe on the right, but on the left there was still extension. This did not change to flexion until eleven days later.

Tournay relates these findings to the general question of agnosia, as well as to the anosognosia of Babinski—the failure of some persons with left hemiplegia with associated sensory disorders to pay any attention to their paralyzed arm.

He also emphasizes the asymmetrical disappearance in his case of both a sensory (agnosia) and a motor (Babinski sign) phenomenon, and the fact that these first disappeared on the same side—the right.

KRAUS, New York.

NEURORECURRENCES FOLLOWING TREATMENT WITH ARSPHEN-AMIN. ERNEST L. ZIMMERMANN, *Arch. Dermat. & Syph.* **5**:723 (July) 1922.

The author studied thirty-nine cases of primary and early secondary syphilis in which neurorecurrences developed during the course of treatment with arsphenamin. This number represents an incidence of 1.64 per cent. In every instance there was a lapse in the treatment, usually from four to eight weeks before the development of a neurorecurrence.

The symptoms of a neurorecurrence are divided into three groups: (1) acute syphilitic meningitis, with or without focal lesions; (2) meningitis of moderate or slight intensity, manifesting itself chiefly by headaches and focal lesions, and (3) no general symptoms of meningitis, the condition being due to a focal lesion. The facial and auditory nerves were found to be involved most frequently.

The author accepts Ehrlich's explanation of neurorecurrence, based on the theory of a sterilization which just falls short of being complete, the host failing to develop resistance against the spirochetes which survive in isolated foci. The pathology is based by the writer on the study of P. Pirilla, who found a diffuse meningovascular process, and on the study of J. H. Lloyd,

who found a localized lymphocytic infiltration. Clinically, the spinal fluid is abnormal in the former; in the latter, it may show marked abnormalities or may be entirely normal.

Only two of the patients had used inunctions to a considerable extent; no patient who had persisted in the course of mercury following arsphenamin developed precocious neurosyphilis. The prevention of neurosyphilis is thus dependent on thorough mercurial treatment.

VONDERAHE, Philadelphia.

AN UNUSUAL CALCIFICATION IN THE BRAIN. WEIMANN, *Monatschr. f. Psychiat. u. Neurol.* 50:202 (Oct.) 1921.

The author gives a detailed study of a case of so-called "calcification of intracerebral vessels." A few cases are reported in the literature, all with the same essential features. Throughout the brain, involving both hemispheres and reaching as far down as the pons, is a widespread deposit of calcium salts. The primary deposit appears in the perivascular lymph spaces. From here it gradually invades the vessel wall, involving first the adventitia, then the media, and finally the intima. When the process is extreme, the whole vessel is converted into a rigid tube, which, on section, stands out above the surrounding tissues. The calcium appears as small concretions or as a finely granular deposit, and if many adjacent capillaries are involved, the whole parenchyma may be filled with coalescing calcium masses. Microchemical studies show that the calcium is deposited in a colloidal (protein) groundwork. The picture differs entirely from that of a generalized arteriosclerosis. The sites of election are the centrum semiovale, the basal ganglia, and the cerebellum. In none of the reported cases has the process extended below the level of the medulla.

Calcification of the intracerebral vessels is a secondary process. It has been observed in various types of local and diffuse degeneration of brain tissue, congenital and acquired idiocy, epilepsy, general paresis, focal softening, etc. In almost all cases, the findings suggest a marked disturbance of lymph circulation. This is probably the primary factor determining the deposition of the calcium salts. Secondary factors depend on the chemistry of blood and tissue fluids.

The clinical picture is not characteristic. It depends on the essential nature of the degenerative process and varies in the different types of cases.

SELLING, Portland, Ore.

PITUITRIN AS A MUSCULAR TONIC. AN ERGOGRAPHIC STUDY. URECHIA and GRAFF, *Ann. de med.* 12:64 (July) 1922.

Struck by the influence of pituitrin on the asthenia of diabetes insipidus, the authors made more ample investigations regarding its action, and have carried out experiments using patients with optostriate or optopeduncular lesions. In epidemic encephalitis, in paralysis agitans and in diabetes insipidus the asthenia is a disturbance of tonus, and in all of these affections neither the suprarenal nor hypophysis presents important lesions which can explain the asthenia.

They employed pituitrin, injecting it in doses varying from 0.01 to 0.02 or 0.03 gm., making ergographic and kymographic curves in thirteen patients before the patients had received pituitrin injections in series as medication, and after such series. The curves were made every half hour during the four hours following an injection. They left intervals, some as long as twenty-three days,

between repetitions of the test in the same patient in order to avoid the misleading effects that "training" might produce. Their tabulations give the number of movements of the weight before exhaustion, and the number per minute, also the distance and the work (weight times the distance), the time before exhaustion and the force (namely the weight times the distance divided by the time). In almost all instances, the curves obtained showed an increasing work and force, presumably due to the injections.

Pituitrin also showed incidentally a marked affect on hypersomnia, especially in one case. This patient, who went to sleep in his chair in the laboratory, could not sleep after the pituitrin injection, sometimes for three to four hours, sometimes for a day. When the injections were stopped, the hypersomnia reappeared, and then disappeared anew when pituitrin was resumed. But it is fortunately only on pathologic somnolence that pituitrin has an influence.

DAVIS, New York.

THE FUNDAMENTAL PLAN OF THE VERTEBRATE BRAIN. B. F. KINGSBURY, *Jour. Comp. Neurol.* **34**:461, 1922.

The longitudinal zones of the early neural tube as defined by His are reexamined in their relations with the plan of the neural plate in still younger stages and with the morphology of the head as a whole. The neural plate ends anteriorly at the chiasma ridge (Johnston) and the primordia on the neural plate of the two optic vesicles are connected across the median plane by a primitive optic furrow just spinalward of this ridge. The anterior end of the floorplate of His is at the fovea isthmi immediately dorsally to the rostral end of the notochord. In front of this boundary the two basal plates fuse in the median plane as far forward as the mammillary recess. Still further forward the two alar plates fuse medially as far forward as the preoptic recess. These alar plates undergo precocious development anteriorly in higher vertebrates and give rise to the whole of the cerebral hemispheres. Their secondary fusion rostrally of the chiasma ridge closes the anterior neuropore, thus forming the lamina terminalis.

These conclusions agree in the main with the well-known views of Johnston and require considerable revision of other current conceptions of the relations of the zones of His in the forebrain. The sulcus limitans is believed to end in the mammillary recess, no part of the primitive motor zones of the neural tube lying rostrally to this region.

C. J. HERRICK, Chicago.

RECOVERY FROM TUBERCULOUS MENINGITIS. A. CRAMER and G. BICKEL, *Ann. de méd.* **12**:226 (Sept.) 1922.

Tuberculous meningitis is generally regarded as fatal, and recovery is so rare that the accuracy of the diagnosis is always questioned.

In this paper, the authors perform a distinct service. They have collected reports of forty-six cases of tuberculous meningitis in which the patient recovered and in which the diagnosis could not be questioned, and from this material an attempt to determine the conditions permitting recovery is made.

Tuberculous meningitis is almost always a complication of tuberculous infection elsewhere in the body. In early life, before the body defenses against tuberculosis have had an opportunity to develop, tuberculous meningitis is almost invariably fatal. The authors found that only one patient under 2 years of age recovered and only six between the ages of 2 and 5. Thus, of

all who recovered, only 17.5 per cent. were less than 5 years old. (When we consider that the great majority of cases of tuberculous meningitis occur in early life and that in children under 12 years of age 83 per cent. of the cases develop before the fifth year—the factor of individual resistance to the infection is of great importance.)

Of the other patients who recovered, 25 per cent. were from 5 to 10 years of age, 2.5 per cent. from 10 to 20 years and 30 per cent. over 20 years.

Among other factors favoring recovery are absence of hypertension of the spinal fluid and low virulence of the causative organisms.

Several methods of treatment reported as contributing toward recovery are mentioned by the authors, but it is their belief that the reason for recovery lies in the resistance of the patient.

Finally, it must not be forgotten that in about 25 per cent. of reported recoveries, there was, in time, a fatal recurrence of the infection.

HYSLOP, New York.

STUDIES IN TRAUMATIC FRACTURES OF THE CRANIAL BONES:

I. EDEMA OF THE BRAIN. II. BRUISES OF THE BRAIN. C. W. APPELBACH, Arch. Surg. 4:434 (March) 1922.

The author notes that cerebral edema has not received the attention given to other sequelae following fracture of the cranial bones. The water content of twenty-six brains removed from persons who died of linear fractures of the cranial bones and associated brain injuries was determined, using the method devised by Waldemar Koch and modified by Mathilde Koch. The water content of six control brains was also studied. The actual increase of water for the entire brain when edema was present varied between 3 and 45 gm.

In the twenty-six cases of the author a division of the bruises of the brain was made into two groups: those which result from laceration of the brain tissue at the time the cranial bones are fractured, and those caused by hemorrhage from torn pial arteries at or near the junction of the gray and white matter. The latter group is made the object of particular study. The brains were examined after the leptomeninges were removed. The bleeding was noted as taking its origin at the junction of the gray and white matter and proceeding to the outside of the brain. The author suggests that gray matter, being more cellular, favors the spread of bleeding.

VONDERAHE, Philadelphia.

THE CASTES OF TERMOPSIS. CAROLINE BURLING THOMPSON, J. Morphol. 36:495 (Sept. 20) 1922.

The two species *angusticollis* and *nevadensis* of the genus *Termopsis* are found on the Pacific slope and in the northwestern United States. The habitat is the decaying wood of forests, rarely in buildings, and never in the earth. Four stable castes are of common occurrence; the first, second and third forms are the fertile reproductive castes; among the soldiers the females are sterile; the males are probably also sterile, though near fertility. There is no true sterile worker class. The sexes are differentiated externally in all castes. Three additional types are of occasional occurrence, second and third forms with small wing vestiges and soldiers with wing vestiges. There is no corre-

lation between the presence of wing vestiges and fertility, but brain size and fertility are invariably correlated. A platelike, nonfunctional frontal gland, without fontanel, is present in first-form nymphs and adults. Vestiges of the lateral ocelli are found in all the castes, except, possibly in the third form. There is a great variability in all organs, and even in the degree of infertility of some female soldiers. *Termopsis* is considered a very primitive genus on account of its many ancestral characters and its close resemblance to the even more primitive genus *Archotermopsis*. The castes of termites are regarded as segregants, arising by mendelian inheritance from a heterozygous parent form.

WYMAN, Boston.

THE CUTANEOUS WHITE LINE (OF SERGENT), SO-CALLED SUPRARENAL. SÉZARY, Ann. de méd. **11**:403 (May) 1922.

In 1904, Sergent described a sign which he considered characteristic of suprarenal insufficiency, namely, a white line produced by light stroking of the anterior abdominal wall. The present article continues the discussion of the validity of this sign with the following conclusions:

There is only one so-called white cutaneous line, and it is the physiologic white line described by Marey in 1858. It is found among healthy as well as among sick subjects. Whether associated with asthenia or arterial hypotension, this white line has no connection with suprarenal insufficiency. Its frequent occurrence in vigorous persons and its existence in patients in whom necropsy later reveals normal suprarenal glands, do not permit any doubt on this point.

The white line presents various modalities. It is, according to the case, sometimes intense and persistent, sometimes slight, of short duration or inconstant, sometimes absent. This diversity of a vascular reaction without doubt depends on the tonus of smooth muscle fibers and on the sympathetic system.

DAVIS, New York.

THE EFFECT OF THE EXTRACT OF THE POSTERIOR LOBE OF THE PITUITARY ON BASAL METABOLISM IN NORMAL INDIVIDUALS AND IN THOSE WITH ENDOCRINE DISTURBANCES. C. A. MCKINLAY, Arch. Int. Med. **28**:703 (Dec.) 1921.

McKinlay determined the basal metabolic rate in twelve normal persons before and after the subcutaneous injection of the extract of the posterior lobe and pars intermedia of the pituitary. Eleven of the twelve subjects showed an increased basal metabolism, the percentile increase averaging 5. In four cases of hypothyroidism there was a diminution of basal metabolism following the injection of pituitary extract. In four cases of subnormal basal metabolism in which the influence of the thyroid was not suggested, injection was followed by a positive response. In four normal persons to whom thyroxin was given intravenously followed in a week by pituitary extract, there was an increase in basal metabolism of 8 per cent. In four other normal persons who did not receive thyroxin the increase amounted to 6 per cent. The author suggests that pituitary extract is effective in accelerating heat production only in the presence of a normally functioning thyroid gland, and that there is probably a synergic action between pituitary extract and thyroxin.

VONDERAHE, Philadelphia.

POINTS OF VIEW CONCERNING FOREBRAIN MORPHOLOGY IN LOWER VERTEBRATES. NILS HOLMGREN, *Jour. Comp. Neurol.* **34**: 391, 1922.

This paper brings out important new facts relating to the most primitive differentiation of primordia of the cerebral cortex in lower vertebrates. The mammalian cortex has three main divisions: hippocampal cortex, general cortex and pyriform cortex. Hitherto there has been no clear recognition of all of these divisions in any animals lower than reptiles; but Holmgren finds in the chief groups of fishes regions of the forebrain which he regards as points of departure for the differentiation of all of these cortical regions. This comes out most clearly in late embryonic stages of elasmobranchs. These observations open up an entirely new field of inquiry in comparative neurology, and may require considerable recasting of current ideas regarding the functional and morphogenetic factors involved in the separation of the cortical from the more ancient subcortical cerebral centers. And these are basic questions in the consideration of the still unsolved problems of human cortical function.

The paper contains also a review of the forebrains of lower vertebrates from the new point of view with an extended discussion of probable homologies of their parts. In conclusion, the problems of forebrain phylogeny are discussed in the light of comparative anatomy and embryology and of palaeontology.

C. J. HERRICK, Chicago.

DIAGNOSTIC VALUE OF SPINAL FLUID WASSERMANN REACTION.

CESTAN and RISER, *Ann. de méd.* **11**:365 (May) 1922.

Vascular cerebral syphilis with meningeal involvement is often difficult to differentiate from paresis, even after a study of the spinal fluid. The authors report the results of an inquiry into the question of whether the spinal fluid Wassermann test performed quantitatively can aid in the differential diagnosis of certain forms of neurosyphilis. The report is based on the study of eighty-two cases of paresis, cerebral arteritis, syphilitic meningitis, tabes and syphilitic meningomyelitis.

In each case several examinations of the spinal fluid were made, extending frequently over a period of several months. The effect of treatment on the fluid was also studied. In each examination the Wassermann reaction was made with different quantities of fluid. With the other reagents constant, the smallest quantity of spinal fluid yielding a strongly positive reaction was called the positive index.

The authors conclude that it is not possible to make a differential diagnosis of the various forms of neurosyphilis on the intensity of the Wassermann reaction in the spinal fluid.

HYSLOP, New York.

CHANGES OF GOLGI'S APPARATUS IN NERVE CELLS OF THE SPINAL CORD FOLLOWING EXPOSURE TO COLD. C. DA FANO, *J. Nerv. & Ment. Dis.* **53**:353 (May) 1921.

The author studied the spinal cords of white rats exposed from two to twenty-two days to cold outdoor temperature. The supply of food was unlimited, and the animals continued to maintain their normal body temperature except when the cold was made more intense or prolonged. Changes were noted in Golgi's internal apparatus in the nerve cells of the dorsal horns, intermedio-

lateral and middle cell column and in the gray matter surrounding the central canal. No apparent changes were noted in the motor cells of the anterior horn. The characteristic of the change was found to be a tendency of the apparatus to assume a more robust aspect and to occupy a "tight perinuclear situation." In some of the cells canaliculi and spaces appeared in the cytoplasm similar to the trophospongium described by Holmgren. In the cases in which the exposure to cold had been such as to lower considerably the body temperature, disintegration of the apparatus was noted.

VONDERAHE, Philadelphia.

DECEREBRATE RIGIDITY IN ANIMALS AND ITS RECOGNITION
IN MAN. F. M. R. WALSH, *Proc. Roy. Soc. Med.* **15**:41, 1922.

After a brief review of the anatomy and physiology of decerebrate rigidity as defined by the work of Sherrington and Magnus, distinctions are drawn between this state of exaggerated tonus of extensor muscles which oppose gravity in the standing position, incapable of modification by other reflex mechanisms, and the conditions which result from removal of the brain just behind or in front of the thalami. In the latter condition tonus is not altered, and the animal can perform reflexly many complex motor adjustments.

Walshe then points out that the posture of extensor rigidity is not sufficient to establish a diagnosis of decerebrate rigidity in man, as it may be brought about by various lesions. He recognizes the fact that true decerebrate rigidity necessarily arises with the greatest frequency in midbrain lesions but may also be caused by damage to the reflex paths concerned and their control as the result of lesions higher up or in the spinal cord. Most of the cases described in the literature do not conform with the criteria laid down by Sherrington and should be excluded from this category.

SINGER, Chicago.

BLOOD ANALYSES IN CASES OF CATATONIC DEMENTIA PRAECOX. S. UYEMATSU and T. SODA, *J. Nerv. & Ment. Dis.* **53**:367 (May) 1921.

The authors select as the basis of their study catatonic cases of dementia praecox because they form a characteristic group and are readily differentiated. Systemic complications and metabolic disturbances were excluded. Control experiments were made on normal blood, and the nonprotein nitrogenous constituents and sugar were determined in thirty-two cases. None of the constituents showed any constant increase or decrease, but the average limits of the amounts of each constituent varied more widely than in the normal; this is interpreted as suggesting an unstable metabolic activity. In 47 per cent. of the cases blood sugar was increased beyond that of the highest normal reading, while in 75 per cent. of the cases a decrease in uric acid was noted; these patients, however, were inactive.

VONDERAHE, Philadelphia.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Oct. 19, 1922

F. H. PACKARD, M.D., *President, in the Chair*

DEMONSTRATION OF A CASE OF PROGRESSIVE MUSCULAR ATROPHY. DR. J. W. COURTNEY.

The patient whom I am about to show is a striking instance of the scapulohumeral type of progressive muscular atrophy. The disorder made its debut on the right side, fifteen years ago. The patient was first seen by me at the Carney Hospital twelve years ago. He was then given a certain number of subcutaneous injections of strychnin. Since that time his treatment has been nil. The wasting of the right shoulder-girdle and arm is now practically complete. The forearm and hand are untouched.

My sole purpose in bringing this man before you is to afford an ocular demonstration of the slowness of the spinal cord decay, even under the most favorable conditions. The patient is a native of the West Indies, 52 years of age, married and the father of five children. From the beginning of his disorder, practically up to the present time, he has worked uninterruptedly as a cook, either in a private capacity or for railroad construction gangs. Only this autumn the left scapulohumeral region began to show evidence of involvement, and the patient now, for the first time, finds his ability to maintain his family in jeopardy. Already the fibrillary twitching of the left shoulder girdle groups is extreme. The left forearm and hand are intact. There is no atrophy elsewhere.

Patients of this kind seldom continue attendance for long at any one clinic; hence, a definite knowledge of their ultimate history is rarely available.

A CASE OF TRAUMATIC ABSCESS OF THE ANTERIOR CEREBRAL FOSSA. DR. J. W. COURTNEY.

My second case is a striking demonstration of the value of the temperature as a guide to the nature of the pathologic sequelae of head injuries. A boy, aged 7 years, on August 10 of the present year was hit in the face by an automobile. Half an hour after receiving his injuries, he was seen by several members of the staff of the hospital at Clinton, Mass. He was conscious. His face was swollen, both orbital regions were ecchymotic, there was an abrasion and hematoma over the left frontal bone, a small laceration on the chin, a fresh defect in the hard palate, a flattening of the bridge of the nose and a tearing away from the jaw of the upper lip and the nose, thereby exposing certain unerupted teeth. There was considerable hemorrhage. The wounds were immediately explored and carefully dressed, but nothing further was done until Aug. 14 when, under light ether anesthetization, the bridge of the nose was elevated. At this time firm pressure was insufficient to correct the defect in the hard palate, so no sutures were taken. The boy stood the operation well and continued to improve from that time on, showing no paralyses, no disturbance in the fundus oculi and no alterations in the

reflexes. The temperature was the characteristic one of *contusio cerebri*, that is, it alternated from normal to about 101.5 F. during the nineteen days of the patient's stay in the hospital. During this time a roentgen-ray examination revealed a linear fracture extending from the region of the roof of the mouth upward in the median line to the ethmoid region, where it deviated obliquely to the left, involving 3 inches (7.62 cm.) of the left frontal bone.

After leaving the hospital, the boy had a temperature of 99.5 to 100 F. for two weeks. From that time on his temperature was normal until ten days prior to his second admission to the hospital on Oct. 7. He had continued under a nurse's care, but he was up and about and had no symptoms. Rather abruptly on October 4 or 5 he developed a temperature of 102 to 103 F., and on October 6 became drowsy and irritable and complained somewhat of headache. The white cell count was 16,000. Now, to retrace my steps a bit: There was noted on September 23 a mucopurulent, bloodstreaked, discharge from both nostrils; but examination of the nose and transillumination of the sinuses revealed nothing significant. Both fundi were normal at that time. On September 29, examination of the latter disclosed a slight engorgement of the blood vessels, and on October 6, the day before I saw the boy, there was a slight choking of the left disk, with moderate engorgement and tortuosity of the vessels of the right fundus.

I examined the patient at the hospital on October 7. He was perfectly conscious, but flushed and inclined to somnolency. The pupils were widely dilated but equal and sluggish to light and accommodation. The fundus was in statu quo. There was no disfiguration of the face, with the exception of a slight flattening above the left superciliary ridge. There was no paralysis of any degree. The reflexes were unaltered. The nose and ears disclosed nothing abnormal. The heart sounds were clear, but there was a distinct intermittence of the pulse, which had not been noted before.

In making a diagnosis I had clearly in mind the favorable channel afforded by the fracture of the nose for the entry of pathogenic organisms. I was also much impressed by the sudden sharp rise of temperature, after a temperature-free period of many days. On these grounds I assumed the presence of an abscess in the anterior chamber of the skull, but from much discouraging previous experience, I was not enthusiastic about operation. However, I advised interference, if within the next few hours the boy's somnolence deepened. He did, in fact, not only grow more stuporous, but developed a ptosis of the left upper eyelid; so the next day about noon, under light etherization, a transverse incision was made through the left eyebrow, and the sinus was opened. This contained a little mucopurulent material and showed a linear fracture in its floor. Its posterior wall was then cut away in an area about one-quarter inch (6.35 mm.) in diameter. The frontal lobe pressed forward into the opening and did not pulsate. The dura was incised and a probe carefully passed around under it. The probe was then passed along the floor of the skull for a short distance, whereupon there was a gush of thick, very foul-smelling pus to the amount of about 4 ounces (113.4 gm.). At this the frontal lobe was observed to pulsate. Irrigation was then done, a rubber dam drain inserted and the wound closed with catgut.

Following the operation, improvement in the patient's general condition was noted for a time, but within a few hours the temperature began to climb to the regions of 105 and 106 F., and clonic spasms appeared in the right side of the face and right arm. These phenomena continued until death.

about 10:30 the following day. At the moment of death the temperature was 108 F. A culture from the abscess showed no growth after twenty-four hours.

I have observed a number of cases of cerebral abscess of varied etiology. Several of these were evacuated with skill and dispatch. I have yet to see one patient survive the operation.

In the case of progressive muscular atrophy shown by me this evening, as well as in all others presenting chronic decay of the central nervous system, there is much food for reflection. They are tragic reminders of the utter futility of the necessarily empiric therapy we apply to them. How may we expect to be relieved of this embarrassing situation? In my own professional life I have followed the growth of the literature concerning their minute pathology to its present appalling proportions. It is an edifying testimonial to unrelenting and meticulously painstaking industry and to inexhaustible patience. But, unfortunately, as it now stands, it is no more vital to the achievement of a scientific therapy than the tissues whence it was derived. To my way of thinking, the data contained in this enormous literature demand to be assayed, so to speak, by a genuinely scholarly mind of the Huxley order. By this I mean a mind which is utterly free from emotional bias; which possesses so truly the gift of sagacious discernment that it never by any chance confounds differences in kind with differences in degree; which is capable of observing and generalizing phenomena and of expressing with punctilious accuracy their order and sequences; and which, withal, is stimulated, and, at the same time, nicely controlled by a sane and vigorous imagination. An assay made by such a mind is bound to yield a universally intelligible conception, not only of the nature of the morbid agents which effect the decay of the nervous elements involved in the diseases in question, but also of their *modus operandi* at every stage of the process. When it is accomplished, the clinician may hope to come into possession of a scientific program of prophylaxis and treatment.

THE PHYSIOLOGY OF AUTOSUGGESTION. DR. DONALD GREGG.

At this time when "autosuggestion" is receiving wide publicity, the subject may well be given consideration by the physician as well as by the layman. Although the physician may attempt to laugh the matter out of court, the layman continues to say, "But there is something in it just the same."

Suggestion implies the passing of an idea from one to another. Without a "conscious" and an "unconscious" autosuggestion becomes impossible. Dr. Morton Prince, whose book on the "Subconscious," popularized the term, has said that there is no such thing as "the subconscious" as distinguished from "the conscious." Dr. Coué in his booklet supplies no proof. He refers to somnambulism, delirious conduct, unconscious memory and control of the autonomic system, all of which phenomena can be explained without considering the unconscious. Most of Coué's thesis regarding self-mastery centers about the control of physical functions through conscious suggestions that are thought to be absorbed by the unconscious, which, in turn, control the bodily functions and produce health or disease. Such a thesis, if tenable, should be applicable in sickness and in health, in infancy and old age, and in the past as well as in the present development of man. What becomes of the unconscious in mental disease? At what age does it begin to develop, and when in man's development did he acquire an unconscious self? If we simply regard the body as a mechanism which automatically runs along healthily in the great majority of cases unless interfered with by noxious agents from without, or unless its activities are handicapped by malformations or maldevelopments, or its functions inhibited or disturbed by the emotions, have we not an unassailable thesis? Without recourse to the confused and disputed terms of conscious and unconscious, we have already in

the intellect and the emotions a duality of functions that explains the situation. We recognize that the emotions modify our vegetative activities, often contrary to our intellect and will, which have little to do with these primitive functions. Here, then, is the physiologic explanation of "autosuggestion" so called. And he who in functional disease heals the sick or helps himself to health, does so, not through the unconscious, but through the action of the emotions. By eliminating the inhibiting or disturbing actions of the emotions in their multifarious forms, functional health is often regained, and he who would heal himself or others needs to know the physiology of the emotions, and not the pseudopsychology of the unconscious.

DISCUSSION

DR. GEORGE A. WATERMAN: The matter is more complex than the division of these reactions on the basis of emotion and intellectual processes only would imply. It is difficult to explain many of the phenomena which are of every-day occurrence, and one is surprised at the number of phenomena that happen in nonpathologic cases. In the every-day life of many of us we find phenomena which, after lying latent in the subconscious, may rise to the conscious, as in the dream states, for instance. So frequently one will wake up and say he has not dreamed at all, when some sudden sound or seeing some object will cause the dream to flash into the mind. These things in the subconscious have an effect on the activities of the person, though he is entirely unaware of their existence. This was illustrated by a young woman who said she thought she had not dreamed, but whose dream was instantly recalled to her mind by seeing a black evening gown hanging in the closet. She had worn the gown to a ball at which she had seen the man of whom she dreamed.

Hypnotic suggestion given in the sleep state produces mental activity without the patient's knowing the cause. I think there can be little doubt that there are activities beneath the threshold of thinking that may produce activities in the intellect. The terminology should be more definitely settled.

RECENT DEVELOPMENTS IN ELECTRO-DIAGNOSIS. DR. PERCIVAL BAILEY.

This article will be published in full in a future issue of the ARCHIVES.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Oct. 27, 1922

C. H. FRAZIER, M.D., *President, in the Chair*

A CASE OF MYASTHENIA GRAVIS OF SLOW DEVELOPMENT. DRs. W. G. SPILLER and M. G. CHADMAN.

The interesting features of this case are the long duration of the symptoms, dating back fourteen years with gradual progression, the comparatively slight implication of the ocular muscles, which usually are among those most affected in myasthenia gravis, and the results of the creatin-creatinin study by Dr. Jonas.

A woman, aged 41, a Hungarian, in 1908 noticed that if she danced for a few hours her legs would become tired and tremble, but after a short rest they would feel well again. By avoiding undue exercise she had no further

symptoms until six or seven years later, when her voice "broke" after a rather lengthy reading; for a few minutes she was unable to speak, but after a short rest her speech returned. The cause of the dysarthria seemed to be a weakness of the lingual and palatal muscles, so that she was unable to form words. About a year later she first experienced difficulty in swallowing, the food lodging in her throat. She could not take solid food and had to live on liquids for a week. For the next four years there was no progression of the symptoms, the only trouble being inability to speak longer than a few minutes at a time, and a feeling of fatigue in the muscles of mastication toward the end of a meal. Within the last two years she has had more difficulty in swallowing. As a meal progresses her jaws become tired and she is unable to swallow solid food, which causes her to cough, after which the bolus is regurgitated. Liquids never cause difficulty in swallowing. About this time she noticed that her limbs would tire quickly after moderate exertion. After riding in an automobile a short distance she would be unable to get up and walk. If she would write for a short time she would have to stop and rest because of muscular fatigue. She then noticed that her eyes would tire on reading and she would see double. To avoid this she read with one eye covered.

At present she has trouble keeping her eyes open, and the difficulty becomes greater as the day advances. She has to exert a constant effort to keep them open and often sits with the head tilted back and eyelids partly closed. She cannot write a postcard without resting, and has to stop and rest several times during each meal. Walking the length of the ward tires her slightly. Her back feels weak and tired. There is no pain in the affected muscles, only a tired feeling after exertion. The hands, forearms, feet and legs feel numb at times. If her hands get cold they become so numb and stiff that she cannot use them. There are no other subjective sensory disturbances.

Physical Examination.—There was no weakness of the ocular muscles except a slight ptosis of the lids. There was slight weakness of the facial muscles, the forehead being wrinkled poorly, the eyelids closing weakly, and the facial lines being poorly marked on smiling and whistling. The muscle power was fair but repeated effort soon caused feeble response, and the movements then became incoordinate. The reflexes (biceps, triceps, patellar and Achilles) were normal and equal on both sides.

After rest speech was clear, but the patient made an obvious effort to control the tongue, lips and palate. After a short conversation the efforts became pronounced, and syllables were not pronounced distinctly. The tongue often appeared to stick, and the patient had to repeat the attempt to pronounce a syllable.

On swallowing, the bolus of food often caught in the throat, and the patient had to make several attempts to swallow it and often failed, beginning to cough and then regurgitating the bolus.

Eye Examination.—The right palpebral fissure measured 7.5 mm., the left 7 mm., becoming slightly narrower as fixation was prolonged. The ocular rotations were full, without diplopia. The media were clear; the disks were slightly oval and healthy large shelving temporal cups. No change in the choroid or retina was noted.

Electrical Examination.—The muscles tested were the orbicularis palpebrarum, orbicularis oris and flexor communis digitorum, the method used being a rapidly interrupted faradic current (tetanizing in effect) which entered the muscles at their respective motor points. A sharp tonic contraction ensued which at the end of about fifteen to thirty seconds began to relax in spite of

the continued excitation. (Comparison was made with a normal muscle in which the current was allowed to flow for five minutes with no sign of relaxation of the tonic contraction.) Repeated galvanic stimulation resulted in sharp responses of a progressively diminishing amplitude of contraction until the muscle would not respond without a rest period. A moderately rapid faradic stimulation (250 per minute) demonstrated failures of contractions.

DISCUSSION

DR. LEON JONAS: During the last decade, there have appeared numerous papers pertaining to biochemical studies in myasthenia gravis. The abnormalities of metabolism centered on three substances: calcium, creatin and creatinin. Certain workers, notably Pemberton and Spiller, found a calcium loss; others found no change in the calcium balance; and a third group reported a calcium retention. A case in which I investigated the metabolism for Dr. Spiller in 1918 showed a slight calcium retention. In view of the conflicting calcium results, it would seem that alterations of calcium metabolism have no particular significance in myasthenia gravis.

Before discussing creatin and creatinin change, I shall briefly explain the normal physiology of these substances. Creatin and its anhydrid creatinin are related to muscle metabolism, and it has been suggested that the urinary output of these substances is in proportion to the muscle bulk. One must be guarded in one's statements on creatin and creatinin metabolism because of the conflicting views on certain phases of this subject.

The creatin coefficient is the number of milligrams of creatinin nitrogen per kilo of body weight. This is approximately 8 in men, but is lower in women. Creatinin is a normal constituent in the urine and is very constant when the subject is on a creatinin-free and a creatin-free diet. Creatin is found in the urine until the eleventh year. It may appear in the urine after this age under the following conditions: first, a low carbohydrate tolerance or insufficient intake of carbohydrate, as in diabetes starvation; second, overstimulation of metabolism as in fever, hyperthyroidism and high protein feeding; third, muscular wastage as in the dystrophies, and when the ability to store creatin is not equal to the supply, as in children. Creatin appears during pregnancy, puerperium and at times during menstruation.

In myasthenia gravis all workers report a low creatinin output. Creatin has been reported by some workers in the urine of patients suffering from this disease. Williams and Dyke found a creatinuria in four cases recently reported in the *Quarterly Journal of Medicine*. Our patient showed no creatinuria. The cases of Williams and Dyke showed a lowered carbohydrate tolerance when tested by giving 100 gm. of glucose and studying the blood sugar curve.

In a normal person, after taking 100 gm. of glucose the blood sugar should not be higher than 150 mg. per 100 c.c. of blood, and sugar should not appear in the urine. The threshold for glucose is 170 mg. per 100 c.c. of blood. In the cases of Williams and Dyke the height of the blood sugar curves were above 170 mg. In the case reported the blood sugar was not more than 123 mg. after giving 100 gm. of glucose, and no glucose was found in the urine.

In view of the association of the impairment of the carbohydrate tolerance in the cases of Williams and Dyke with the presence of creatin, the absence of creatinuria in our case may have some relationship with the normal or rather increased tolerance for glucose.

DR. FRANCIS X. DERCUM: Before the patient departs I would like to call attention to the nasal intonation of the patient's speech due to the involve-

ment of the soft palate and also the marked accentuation of both nasolabial folds which gives an expression suggesting the risus sardonicus. In my experience this deepening of the facial folds gives an almost characteristic facial expression to patients with myasthenia gravis.

THE ASSOCIATION OF HERPES ZOSTER AND TABES DORSALIS.

DR. W. B. CADWALADER.

The occurrence of herpes zoster in tabetic patients or in patients suffering from cerebrospinal syphilis does not seem to be common. Head and Campbell (*Brain*, No. 3, 1900, p. 366) found that the alterations that take place in the posterior spinal ganglions and in the gasserian ganglion of patients with herpes zoster of syphilitic origin resemble those found in other conditions. These changes are: (1) inflammation with exudation of small deeply staining cells, (2) extravasation of the blood, (3) destruction of ganglion cells and fibers, and (4) inflammation of the sheath of the ganglions. The authors state that in tabes dorsalis outbursts of zoster form a classical symptom, but that this phenomenon is rarer than textbooks would lead one to believe. They also state that paresis predisposes to zoster, and they were inclined to attribute it to the arterial changes that take place in the ganglions and not necessarily to the primary syphilitic process.

The involvement of the sympathetic nerves to the eye causing sympathetic ophthalmoplegia that occurred in my second case can be attributed to the syphilitic process affecting the sympathetic nerves that pass close to the ophthalmic division of the trifacial nerves in its course to the gasserian ganglion. Dr. Dorrance, in a paper on alcoholic injections of the gasserian ganglion, presented before this Society a few years ago, called attention to the fact that this produced flushing of the face and lachrymation—symptoms of sympathetic involvement. The severe pain in the eyeball with lachrymation and photophobia that also occurred in my second case greatly resembled the so-called "optic crises of tabes." Dr. Spiller made a special report of such a case before this Society a number of years ago.

CASE 1.—A woman, 60 years old, during the summer of 1921 developed considerable pain in the right upper limb, quickly followed by a typical herpetic eruption on the outer side of the upper arm and of the forearm. Soon after this she began to have lancinating pains in the lower limbs followed by diplopia and by ataxia in walking, and also girdle sensations.

Physical examination revealed the usual signs of tabes dorsalis, and Dr. deSchweinitz reported that both fundi were normal. The Wassermann reaction was positive.

CASE 2.—A man, 65 years old, was healthy until July, 1922, when he suddenly developed severe burning pain in the left eyeball with much lachrymation. The pain later extended through the left side of the nose and over the left forehead. After a few days had elapsed, a typical herpetic eruption appeared throughout the distribution of the ophthalmic division of the fifth nerve. Except for these symptoms, he felt fairly well.

Physical Examination.—The right pupil was small and reacted slowly to light and in accommodation. Ocular movements were normal. The pupil of the left eye was contracted, and the left palpebral fissure was narrower than the right. The left cornea was anesthetic; there was hyperalgesia throughout the distribution of the ophthalmic division of the fifth nerve on the left side. In this distribution scars of the herpetic eruption were still present. Pain, though

not nearly so severe as formerly, had not disappeared. Dr. deSchweinitz reported that the patient had left enophthalmos and slight myosis unaffected by cocaine. There were no fundus lesions, and the fields were normal. The gait and station were normal; tendon reflexes of the lower extremities were greatly diminished but not lost. The Wassermann reaction of the blood serum was negative. Sensation over the distal portions of the lower limbs for pin prick was slightly delayed; over the thorax it was normal.

DISCUSSION

DR. SPILLER: In 1893 or 1894 I saw a woman who had cervical Pott's disease. She had herpes zoster in the distribution of the second thoracic root only on one side. At the necropsy the first thoracic root was removed by mistake for the second, and it was entirely normal. The second thoracic root when removed was found to be covered with miliary tubercles. This is the first time to my knowledge that a distinct relation of herpes zoster to disease of the spinal ganglion has been established.

DR. CHARLES H. FRAZIER: Frequently after operations on the gasserian ganglion herpetic eruptions occur, possibly one in every three cases, but so far as I remember I do not recall having seen any in the first division. The great majority were in the second division, the others in the third; they were almost invariably on the lips.

AN ACUTE ALCOHOLIC PSYCHOSIS ASSOCIATED WITH BRAIN TUMOR. DR. C. W. BURR.

A white man, 51 years old, went to the Philadelphia Hospital on Sept. 5, 1922, complaining of headache. There was a superficial wound 2 inches (5.08 cm.) in length above the right temple. He said that he had been hit with a hammer, but he was so confused that he could not give any details of the injury. He said that he had been drinking hard for two weeks. When put to bed he became more confused, picked at the bed clothes, rocked back and forth and snapped his fingers. He seemed to have particular difficulty in recalling words. He was left-handed. A few days later a relative was found who said that the patient had been a wanderer for the last few years, previous to which he had been a professional ball player. His habits previous to leaving home had been good, and the relative said that he had always acted normally, although she had not seen him for several years.

Physical Examination.—The restlessness present on coming to the hospital decreased, but the man continued to be dazed. When asked a question he would stare at the questioner and repeat the words several times. Sometimes he would finally give an answer, usually relevant; sometimes he would not reply. There was no paralysis of the arms, legs, face or tongue. He had no difficulty in swallowing and no disturbance of articulation, but quite frequently he was unable to recall a word. His abdominal and thoracic organs were normal. There was no disturbance of any of the reflexes, and the gait was normal. He was careless about his bladder and rectum and unconcerned about his habits. At times he was irritable, at times quiet. He adopted several muscle habits, for example, he would stir an empty pan with a spoon for many minutes at a time and would rub a dish back and forth on the table. On October 12 he had a general epileptiform convulsion, after which he slowly sank into coma and died eight days later, having been in a coma throughout that entire period. Both the blood and spinal fluid Was-

sermann reactions were negative. Roentgen-ray examination of the head showed no fracture. Dr. Langdon reported that the pupils responded well to light and accommodation, that the ocular rotations were full and equal, and that there was no nystagmus. The ocular arteries were small, but there were no signs suggesting sclerosis. The disks showed some loss of capillarity without other fundus changes.

During the first two weeks his temperature was a little subnormal, during the following four weeks it was normal, and during the balance of the time it ranged from 99 to 102.5 F.

At necropsy a glioma of the right frontal lobe was found and in addition, chronic myocarditis, chronic endocarditis, splenitis, interstitial nephritis and chronic perihepatitis. The tumor mass had no wall, infiltrated the nerve matter, and there was a hemorrhage within its degenerated center.

I doubt if the tumor caused the symptoms. The mental picture was that of chronic cerebral alcoholic poisoning. Headache was the only symptom of tumor of which he complained, and there were no physical signs of it. The one convulsion may have occurred at the time of the hemorrhage.

THE AVELIS SYNDROME. DR. ALFRED GORDON.

A man, 38 years of age, became ill about three months ago, while getting off a car, when he noticed that he inclined toward the left when walking, although this symptom lasted only a few minutes. Several days later he suddenly developed severe headache in the right temporoparietal region, which persisted several hours unaccompanied by vomiting or dizziness. The next morning he felt very weak and could not go to work. The headache reappeared on the same side and was accompanied by vertigo, both lasting from five to ten minutes. This was followed immediately by difficulty in swallowing and awkwardness in the left upper limb. At the end of five weeks he presented the following condition: The gait and station with eyes open or closed were normal. The movements of the extremities were correct, and the grip of both hands was normal. The head was slightly tilted to the right, and the face seemed slightly deviated to the left. The right orbital fissure was wider than the left. The pupils were equal and reacted to light, but the right responded more sluggishly than the left. The ocular muscles and eyegrounds were normal.

There was no ataxia, adiadokokinesia or tremor in the upper extremities. The knee jerks were markedly increased; there was no ankle clonus, and the plantar reflexes were of the flexor type.

Sensation was altered on the left side of the face (in the distribution of the second and third branches of the fifth nerve), neck, thorax as low as the mammillary line, and on the left shoulder, arm and hand; in this area the sense of touch was normal, but pain and temperature perceptions were considerably diminished, cold being taken for warm, and hot was in some places not recognized at all. The deep sensibility on the left side was not involved. Another conspicuous symptom was the inability to swallow liquid or solid food, so that the patient had to be fed through a nasal tube. The soft palate and uvula, the epiglottis and entire larynx appeared drawn to the left. The vocal chords functioned normally; sensation over the right side of the soft palate and uvula was greatly diminished. The sense of taste, the tongue and hearing were normal. A vestibular test showed loss of function of the vestibulo-ocular as well as of the vestibulo-cerebello-cerebral pathway from the vertical semicircular canals of both sides. The horizontal canal

pathway appeared to be unaffected. The Wassermann reaction of the blood and spinal fluid were normal. His mentality was good. Urinalysis was negative. The heart was somewhat enlarged, and a mild presystolic murmur was present at the base.

Repeated examinations of the patient invariably gave the same results. Only a few days ago some modifications were observed for the first time. The syringomyelic sensory dissociation described in the foregoing became less marked on the left side of the face. The area of the second and third branches of the left trigeminus became less affected; instead of complete loss of pain and temperature perception, there was merely a slight diminution of these two sensibilities, and the real loss of sensations commenced at the lower border of the mandible. Moreover, the deviation of the face to the left was also less marked than before. The patient had recently observed that small quantities of fluid passed down the esophagus without much difficulty.

The most conspicuous symptoms in this case were the difficulty in swallowing and the sensory dissociation on the face and left arm and thorax. The lesion evidently lay in the medulla, involving the nuclei of the ninth, tenth and seventh nerves, also Gowers' tract. The lesion was evidently vascular and consisted of an occlusion of the posterior inferior cerebellar artery. The case is interesting from the standpoint of the limited number of symptoms, which is contrary to what is found in the majority of cases of this character.

DISCUSSION

DR. W. B. CADWALADER: The distribution of sensory disturbances in Dr. Gordon's patient appear to be most unusual, for the anesthesia was on the face and limbs of the side opposite to the lesion. Usually in cases of thrombosis of the posterior inferior cerebellar artery there is disturbance of sensation of the face on the same side as the lesion in the medulla oblongata, with disturbance of sensation of the limbs on the opposite side.

DR. SPILLER: The explanation of the symptoms in Dr. Gordon's case, it seems to me, is to be found in a lesion of the right side of the medulla oblongata, the side on which the paralysis of the soft palate occurred. The disturbance of pain and temperature sensations on the left side of the face is to be explained by implication of the central fibers passing from the nucleus of the left spinal root of the fifth nerve and after decussation ascending on the right side of the medulla oblongata. The limited implication of pain and temperature sensations on the left side of the trunk extending only a short distance downward indicates that the fibers in the spinothalamic tract from only the part in connection with this distribution were implicated on the right side.

CLINICAL AND PATHOLOGIC DIFFERENTIATION OF THE SENILE PSYCHOSES AND THE ARTERIOSCLEROTIC PSYCHOSES. DR. A. R. VONDERAHE.

The psychoses peculiar to old age, according to the current classification, are divided into two groups: the senile psychoses and the arteriosclerotic psychoses. Some clinicians do not follow this grouping but consider the term "senile dementia" adequate for both types; others include both under such a term as "arteriosclerosis of the nerve centers."

A separation into senile psychoses and arteriosclerotic psychoses, however, can be made on the basis of clinical symptomatology. An analysis of eight

consecutive cases from the Pennsylvania Hospital was made. Seven of these cases were classed as senile psychosis; one as arteriosclerotic psychosis. The differentiation rests on excluding the symptoms characteristic of cerebral arteriosclerosis, such as headaches, dizziness, feelings of pressure, syncopal attacks, and particularly the finding of some focal sign referable to an arteriosclerotic process. In the seven cases diagnosed as senile psychosis the mental symptoms were found to be much the same in all: impairment of memory, disorientation, confusion. In addition, in one case there were visual and tactile hallucinations; in another a state of apprehension with delusions of infidelity, and in another a tendency to maniacal attacks. Physical examination in each instance showed evidence of cardiac impairment, either hypertrophy, irregularity or a murmur or a combination of these. In all the cases diagnosed as senile psychosis there was sclerosis of both radial and temporal arteries with tortuosity; in two instances there were unequal pupils; in one instance the pupils were irregular and did not react to light; in one instance, in which the eyegrounds were examined, arteriosclerosis and tortuosity of the retinal vessels were found. The blood pressure was not constantly increased. In every instance but one the urine contained albumin and hyaline or granular casts. In the cases which were diagnosed as senile psychosis, accordingly, renal and myocardial changes were found to be prominent in the symptomatology.

The views of Kraepelin, Bing, Alzheimer, Nötzli, Meyer and Appledorn regarding the gross and microscopic pathology of the condition, vary. Southard had hoped to establish a case of pure senile atrophy but was not able to exclude arteriosclerosis entirely in any one instance.

The available pathologic material and clinical records at the Philadelphia General Hospital were studied. In a total of 435 brains, 189 showed arteriosclerotic changes. Of the total which showed arteriosclerosis, only twenty-five, or 13.2 per cent., were diagnosed clinically as senile dementia, thus illustrating the well-known observation that marked arteriosclerosis may occur in the cerebral vessels without psychosis.

On the basis of the differential points noted in the foregoing, the cases were grouped into senile psychoses and psychoses with cerebral arteriosclerosis. The gross and microscopic anatomy was studied. In all cases but one evidence of cerebral arteriosclerosis was evident grossly and microscopically. In the latter case, however, microscopic examination of the frontal, occipital, parietal and temporal cortex showed thickening of a patchy type in the smaller vessels, while the larger vessels apparently escaped; there was also thickening of the pia, some glial proliferation and evidence of retrograde processes in many of the cortical cells. The systemic pathology of this case is of interest. There was ulcerative arteriosclerosis of the aorta, sclerosis and calcification of the coronary arteries with myocardial degeneration. The kidney grossly showed evidence of chronic passive congestion and microscopically, arteriosclerosis and toxic changes. Clinically, the psychosis developed about a year before death and was characterized chiefly by incoherent speech and depression. On physical examination the heart was found to be enlarged to the left; there was a harsh systolic murmur at the apex, transmitted to the axilla and an occasional extrasystole.

Seven of the cases could be classed as senile psychoses. One of these cases has just been described. In the remaining six, cortical atrophy, pial thickening and gross arteriosclerosis of the basal vessels were found. Microscopically these brains showed cellular atrophy, diminution of the cells of

the outer layers of the cortex, increased glial proliferation, evidence of retrograde processes and lipoid accumulation in one instance. Varying degrees of sclerosis of the smaller arteries were noted.

Twenty-five cases diagnosed as "senile dementia" from the Norristown necropsy reports were analyzed. In two instances in which the brain was described but no mention made of the arteries the visceral changes were noted in detail; in one there was found chronic aortic and mitral stenosis, coronary arteriosclerosis, chronic interstitial nephritis and cirrhosis of the liver; in the other, chronic interstitial myocarditis, mitral and aortic valvulitis, chronic atrophic gastritis, chronic cholecystitis, cirrhosis of the liver and chronic interstitial nephritis. From the same source it is found that eighty-four of ninety patients with senile dementia have nephritis in some form or other, sixty-two having chronic interstitial nephritis, seventeen chronic parenchymatous or chronic diffuse nephritis, and five suppurative nephritis.

In conclusion, it may be noted that the term senile dementia is sometimes used to include the senile psychoses and arteriosclerotic psychoses, but that a clinical separation into two such forms can be made and is useful. Pathologically, there is difficulty in separating the two. In cases diagnosed as senile psychosis, the consistent presence both clinically and pathologically of recognizable heart or kidney lesions is emphasized. Cases of senile atrophy with moderate arterial change, or at least with arterial change insufficient to satisfy the observer of a casual relationship to the atrophy, have been described. In such cases, the rôle of arteriosclerosis and other pathologic conditions in the viscera is suggested as forming a possible basis for a toxic neuronc degeneration of the cortex.

DISCUSSION

DR. CHARLES K. MILLS: As has been pointed out, in senile dementia, as recognized both clinically and pathologically, just as in cases which are definitely to be designated as cerebral arteriosclerosis, vascular disease is present. That fact was notable in some of the observations quoted by Dr. Meyer and others.

Two points seem to help most in the differential diagnosis of these affections. They depend on the question of the degree of the vascular disease and on the question of the locality of the lesion which is present. Many of the symptoms of senile dementia, as seen in both hospital and private practice, are due, not to destructive focal disease, but are dependent on the lack of a proper blood supply passing through vessels that are not sufficient in caliber normally to nourish the brain. Consequently, there is a general depression in cerebral function and a change in the mental phenomena presented by these patients. We get not only impairment of memory, both in arteriosclerosis, so-called, and in senile dementia, and perhaps a certain degree of disorientation, but in true senile dementia recognized as this psychosis, we have mental symptoms of peculiar type, some of which have been mentioned. We have hallucinations of particular type, and we have also what seems to me to be of particular importance for true senile dementia, a peculiar form of persecutory delusions—a sort of reversal of ordinary familial relations. In old age, in cases which are properly called senile dementia, the person suffering from this affection will turn from the members of his family who have always cared for him and helped him in every way, perhaps to some member of the family who has not taken any interest in him, or has even neglected him.

I have seen many, and some sad, cases of cerebral arteriosclerosis. I have had an opportunity to study them in private practice over a series of years, cases which have developed before a really general senile condition had arisen. The one thing that has led me to a positive diagnosis of cerebral arteriosclerosis, having in the first place excluded certain focal lesions like brain tumor and focalized meningitis, is the fact that there are symptoms of mental dissociation which are evidently due to focal lesions, especially in the deeper regions of the brain. After loss of memory, there is disorientation, often then apraxia, and next aphasia of the dissociating type. This aphasia becomes more marked until verbal incoherence is present. In my own experience, headache has not been so prominent a symptom. Curiously, in some cases patients who have suffered from extremely severe recurring sick headache, after the development of clear indications of cerebral arteriosclerosis, have had a diminution in the severity and in the frequency of the headaches. Headaches are, however, sometimes present, but I would not give them the importance assigned by the reader of the paper.

Areas of multiple softening will dissociate eventually one part of the brain from the other until the entire brain is dissociated, whereas in the cases of senile dementia, there is a general parenchymatous or nutritive disorder of the brain which is due to an insufficient blood supply, but which is not so frequently accompanied by focal lesions in connection with vascular disease.

LEFT HEMIPLEGIA WITH APHASIA IN A LEFT-HANDED PERSON,
AND WITH AGRAPHIA IN THE OTHERWISE UNAFFECTED
HAND. DR. ALBERT C. BUCKLEY.

The following case report is of unusual interest and therefore worthy of record, because it deals with an aphasic and agraphic left-sided hemiplegic patient who was naturally left-handed. She had educated the right hand for writing, but had lost the ability to write with the right hand when the left side became paralyzed.

The motor aphasia was at first complete; later marked improvement in speech followed, but the ability to write, up to the present time, nearly six months after the stroke, has not returned. The patient was naturally left-handed, that is, she always took the initiative with the left hand to grasp objects, and she sewed with the left hand. She had learned to knit and to write, however, with the right hand, and always used the right hand for writing.

The early medical history presents nothing of importance from the standpoint of the present illness. She had suffered from severe attacks of "indigestion," and was treated for gastric ulcer in 1904 and in 1908, from which she apparently recovered.

The course of the patient's disorder is as follows: While occupied with household duties, the patient was seized with a feeling of fatigue, difficulty in holding objects with her left hand, loss of speech, followed by total loss of power in the left hand, and finally paralysis in the left leg, in the order named. Physical examination revealed a large framed, middle-aged woman, who presented the general appearance of a person in robust health, weighing about 200 pounds (90.72 kg.). The pupils were unequal, the right larger and flattened on the superior margin. There was prompt iris reaction in both eyes to light stimulation and with convergence; consensual response was preserved in both eyes. There was no hemianopsia. There were no marked changes in the fundi; the disks were described as being paler than is usual for a

person of her age; the arteries were distinctly thickened and their caliber reduced. There was no ocular palsy, no weakness of the ocular muscles or nystagmus.

There were no other cranial nerve signs than the apparent weakness of the tongue causing it to deviate slightly to the left of the midline. The left side of the face was slightly flatter at the cheek, and distinct weakness of the same side was shown when the mouth was opened wide. The tongue deviated slightly to the left of the midline, but was protruded without weakness or tremor. The throat was negative although swallowing was difficult.

There was a total, flaccid, left-sided hemiplegia and hemianesthesia. The patient was unable to move in bed, lying on the left side by preference on account of pain in the region of the left flank. Passive movements of the paralyzed arm caused pain in the shoulder region of that side. The biceps jerks were present on both sides, those of the paralyzed side being more active. Both patellar reflexes were active, the left more than the right. The plantar reflexes were present on both sides; stroking the right sole produced the normal plantar flexion; on the left side there was a definite Babinski reaction.

All forms of sensibility were lost on the paralyzed side, except occasional pain reactions to deep pricks. Faradic stimulation caused movements of the arm and leg muscles with the production of painful sensations in the paralyzed limbs.

There was no disturbance of superficial or deep sensibility on the right side; objects placed in the hand were named promptly when the patient could find the word, and when not able to find the word she was able to indicate by pantomime the use of the object. From this it is inferred that there was no stereognostic disturbance and no apraxia on the right side and no disturbance of sensibility. Thus far the patient presented the physical signs of a left-sided hemiplegia with anesthesia, the right side being functionally intact from the standpoint of sensibility and motility. There was at first complete incontinence of the bowels and bladder, due to mental dulness. The patient was at first practically wordless, with the exception of an occasional word which was distinctly enunciated, as most of her attempts at word formation were either unintelligible or sounded like "baby talk" whenever spontaneous speech was attempted. Words pronounced for her could, however, be repeated by the patient at the time. Attempts to combine words she had just pronounced invariably resulted in failure.

There was no word deafness, as was shown by her ability to pronounce some words given her and her understanding of most of what was said to her. She was interested in stories read to her and later was able to convey in part, by gesture and a few words, the meaning of what had been read.

She followed commands, as already indicated, and carried them out. Unusual words such as "Oshkosh," and "Kalamazoo" were pronounced with difficulty, after they had been repeated several times, carefully separating the sound of each syllable.

She was not object blind as shown by the fact that she recognized the use of common objects, such as a knife, key and pencil. She clearly indicated that she recognized individual letters and numbers at sight, and could name some of them as digits but failed in compounds. She later was able to count spontaneously from 1 to 16; then said "18, 9, 10, 11." She recognized such numerals as 20 and 30, but when shown 203, said "20 and 3." A few familiar words were recognized and pronounced, for example, "4th of July."

The remarkable disturbance in this patient was the loss of ability to write with the unaffected hand she normally used for writing. The agraphia was complete, including attempts to write her name. At times the agraphia appeared to be due to inability to use the correct word. For example, when told to write "plate" she said "dish" and attempted to write "dish," but failed. She recognized that her writing did not look right and was greatly annoyed by the fact. She did better at writing numerals from dictation, but had difficulty with these. In attempting to write 13, she said "one, one, one, three" and wrote 1113. She was unable to write the names of the days of the week after she had learned to repeat them. Attempts at spontaneous writing were hopeless failures. Writing from copy was also practically impossible, as only an occasional word could be legibly written.

During the first six weeks there was considerable improvement in speech, her vocabulary slowly increasing so that some sentences were completely formed. Words were frequently badly pronounced and often a word could not be found to complete an idea.

The difficulty in swallowing gradually disappeared, but the paralysis, except for slight power of adduction of the leg, was unchanged except that con-

The image shows a handwritten specimen of the patient's writing. The text is written in cursive and reads "It is a bright day." The handwriting is somewhat shaky and shows signs of difficulty, particularly in the spacing and the final letters of the words.

Specimen of patient's writing. "It is a bright day."

tractures appeared. There was little improvement in sensibility. Deep painful stimuli were felt here and there, but the patient was unable to locate the sensation thus produced.

The agraphia persists at the present time, six months after the onset of the disorder, there being no improvement in the attempts to write.

In the literature I have thus far not been able to find a similar case. Claude (1910) reported an instance of complete hemiplegia on the right with apraxia on the left. There was no apraxia in my patient. Pitres (1884) recorded a right-sided paralysis without aphasia in which the patient is reported as unable to write with the right hand, but wrote freely with the left hand. Meyer (1908) reported a left-handed man with left hemiplegia who was unable to write words, who could write letters but not the letters asked for.

One of my left-handed patients, a woman 70 years of age, parietic on the right and astereognostic, had lost her ability to write with the right hand, and later learned easily to write well with the left hand. Another elderly woman, right-handed, with a powerless right hand and marked dysarthria, but not true aphasia, is totally unable to write with the left hand. Another of my patients, now a right-sided hemiplegic, a right-handed person, is not agraphic in the left hand, although she writes with much difficulty.

It is an interesting question whether the patient under discussion, if she had been right-handed would have been agraphic on the right. It might also give valuable data if we were to determine whether our hemiplegic patients show any impairment in original writing ability of the contralateral hand. All of us are able to write legibly to a certain extent with the untrained hand. Is this impaired, and to what extent, after a paralysis on the opposite side?

In attempting to explain the mode of producing the agraphia in this case several possibilities were considered: 1. Bilateral lesions can be reasonably excluded on account of the absence of any physical signs in the right arm or hand. 2. A lesion in the visuopsychic cortical region on the same side as the principal lesion is excluded on account of the absence of mind-blindness. With the exception of failure to read written phrases correctly there was no distinct involvement, except early, when the motor aphasia was so marked that she could not pronounce any word. She read names of objects and designated her recognition of those objects at the same time. 3. A subcortical lesion sufficiently large to include the paths from the motor, the sensory and the visuopsychic areas can also be excluded on similar grounds.

For years we have been following the subject of aphasia with utmost respect for diagrams of hypothetical construction, on which our explanations of speech disturbances in particular were based. More recently came the revisions of the subject by Marie and Bernheim. It seems that much is still to be desired from the standpoint of definite information and data concerning aphasia.

As Monakow stated (1906), it must be admitted that the more closely the clinical phenomena presented by patients with aphasia are studied, and the more accurately the anatomic changes are examined, the greater is the difficulty of furnishing a satisfactory explanation of the connection between the site of the lesion and the symptoms of aphasia in observed cases, and in spite of the persistence or increase in the size of the lesion in the speech region, the aphasic symptoms disappear; in other cases, the lesion may be beyond the limit of the speech region, yet the aphasia may persist for a long time. Paradoxical cases of sensory aphasia with destruction of Broca's area and motor aphasia with lesions in the temporal lobes are as yet unsatisfactorily explained. Monakow would explain many of the foregoing apparent discrepancies on the basis of a temporary suspension of function arising from the local interruption by disturbance of a tract of fibers which directs or carries out the function of a neighboring part. Monakow terms this diaschisis and states that aphasic symptoms are partly those of a more or less temporary character when not due to "neighboring symptoms" or to allied circulatory disturbances and are produced by diaschisis. The aphasia may depend on the diaschisis in distant regions of the brain more than on the lesion itself.

This patient, it is to be emphasized, was by birth and by nature left-handed. The right hemisphere, it is to be inferred, had become functionally developed. Furthermore, this condition developed in a person whose writing center was not brought into use until she learned to write, at least five years after the normal development of the cortex and pathways were completed; in other words, this function of the cortex, in this particular region, was acquired later than most of the coordinate motor functions.

It seems reasonable to conclude that a widespread lesion, as is evidently present in this case, if we are to judge from the total hemiplegia and hemianesthesia, may interfere with cerebral function as a whole (and I am assuming that we cannot restrict the functions of speech to a localizable motor region of the cortex). In fact, the effects of more or less localized lesions on the

entire cerebrum are by no means slight. In this case I believe that the agraphia on the otherwise unaffected side is dependent on the lesion at a distance. The function of writing, which in this case was acquired later than other motor functions, was less stable and therefore more easily affected, perhaps by diaschisis, but certainly by reason of its being comparatively a less organized function.

DISCUSSION

DR. CHARLES K. MILLS: No doubt in this case the right hemisphere primarily was the active engine in speech, writing and perhaps much else. The right hand was trained to do many things which the left hand otherwise would have done. The real ability to write remained in the right hemisphere in a congenitally left-handed patient, and when a large left-sided lesion of the brain occurred, although there had been an apparent transfer, it did not really result; the patient returned, so to speak, to congenital conditions.

DR. FRANCIS X. DERCUM: Dr. Buckley's case calls to mind one that was reported by Byrom Bramwell many years ago, in which the patient first suffered from a lesion of the left side of the brain, became aphasic, subsequently recovered the speech faculty, and then had a second lesion on the right side of the brain. He again became aphasic and remained aphasic permanently. Dr. Buckley's case, like Dr. Bramwell's case, is suggestive of a double rôle played by the two hemispheres in regard to the function of speech, although this function is one that is highly specialized. Dr. Buckley spoke of his patient as recognizing separate letters. She was notwithstanding unable to read, that is, she could not combine the separate letters into words or the words into phrases and sentences: in other words, she had alexia. One of the remarkable clinical facts of aphasia is that in so-called motor aphasia we always have alexia. I have met a motor aphasic who could read. Writing also is a highly specialized faculty and one acquired relatively late, and it would seem to me that the facts in regard to alexia apply also to agraphia.

I am very glad that Dr. Buckley clearly separates the purely dysarthric symptoms from the intellectual defects of aphasia. Further, I think we are too much in the habit of speaking of the specialization of the left hemisphere. The biologic fact is that the left hemisphere is always the larger of the two, it is dynamically the greater of the two and it almost stands to reason that when this greater half is crippled by a lesion, the functions of the right hemisphere are also impaired, that is, they are inhibited; and von Monakow's theory of diaschisis has a direct application here. The acquisition of left-handedness by right-handed persons is of course not unusual. We all of us recall the instance of a distinguished surgeon who was the teacher of many of us who was ambidextrous, and this quality had its origin in the fact that very early in life he suffered a serious injury of the right hand so that the left hand was of necessity trained, and trained very early.

DR. SPILLER: It is generally stated, and sufficient cases are on record to show the truth of the statement, that in right-handed people the speech area is in the left side of the brain, and it is probable that in left-handed people the speech area is in the right side of the brain. There are a few cases recorded in which, in right-handed people, the speech area has seemed to be on the right side of the brain, not on the left. There are cases recorded in which Broca's area on the left side has been destroyed, without disturbance of speech. It is important to determine whether the lesion in such cases developed early in life. It is unusual to find persistent aphasia in a right-handed

child who has a right hemiplegia that developed early in the left, because the right cerebral hemisphere is capable of assuming the speech function when a lesion develops within the first few years of life.

There are people who are ambidextrous, who probably have the speech mechanism in both sides of the brain. It is customary for a person who is left-handed to learn to write with the right hand. He is taught to do this in school.

On one occasion I had reason to believe that a certain man had an abscess of the brain in the left temporal lobe. He had no signs of sensory aphasia, and he was said to be right-handed because he was known to write with his right hand. He did have the abscess in the left temporal lobe, although he did not have sensory aphasia, but he was left-handed, and his child also was left-handed.

It is interesting to see how readily a child may become left-handed. I recall a baby of parents who were right-handed, and no left-handedness was in the family. The child had used the right hand like any right-handed child until this hand was burned. The bandage on the hand made the child use the left hand, and after recovery from the burn of the right hand the child was left-handed and had to be taught to use the right hand again. If he had been allowed to use the left hand, it is possible that the speech area would have developed in the right cerebral hemisphere.

We must assume in Dr. Buckley's patient that the speech mechanism was on the right side of the brain. Agraphia may occur in sensory as well as motor aphasia. I do not believe that if his patient had not been taught to use the right hand for writing she would have been able to write with the right hand after aphasia developed. The peripheral mechanism is under the control of the cortical mechanism. The ability to write with the right hand in his patient probably was controlled from the right hemisphere. A right-handed person with agraphia cannot write with the left hand.

A STUDY OF THE SPINAL ACCESSORY NERVES FROM A CASE OF BILATERAL ACQUIRED SPASMODIC TORTICOLLIS. DR. M. A. BYRNES.

A review of the literature on the pathology of the peripheral nerves in this disease revealed only one instance in which a similar study had been made. Ballance at the conclusion of his article on torticollis merely stated that the nerves showed nothing abnormal. Southam had, however, examined a portion of the spinal accessory nerve which had been previously subjected to stretching. This nerve showed slight pathologic changes which were, no doubt, due to injury in the earlier operation, and Kadner studied the intramuscular nerve fibers in congenital torticollis, but failed to find any marked changes.

In the case reported, a portion of each spinal accessory nerve and the posterior muscles on both sides of the neck were preserved in formaldehyd. The nerve was stained in cross and longitudinal sections by the following methods: hemalum and acid fuchsin, Weigert, van Gieson and phosphotungstic acid. The muscle tissue was stained with iron hematoxylin and hematoxylin and eosin. The Weigert preparation of the nerve showed almost total disintegration of myelin. In both the cross and longitudinal sections stained with phosphotungstic acid, there was definite fragmentation of the axis cylinders. The muscle tissue showed the changes which usually have been observed in previously studied cases of this affection. So far as I have been able to determine, this is the first instance in which changes have been described in

the peripheral nerves in acquired spasmodic torticollis. I do not feel, however, that any conclusion as to the etiology or significance of this pathologic finding can be deduced from this single observation, nor am I of the opinion that these changes discredit the existence of a mental torticollis, although it is my belief that in this particular case the condition was of organic origin.

DISCUSSION

DR. ALFRED GORDON: Several years ago, with Dr. Rowe, an aural surgeon, I investigated pathologically the subject of so-called neuralgia. We examined the extirpated nerves in several cases of facial neuralgia and found some degenerative changes.

Book Reviews

THE ELEMENTS OF SCIENTIFIC PSYCHOLOGY. By KNIGHT DUNLAP, Professor of Experimental Psychology in the Johns Hopkins University, Baltimore; Author of "Mysticism, Freudianism and Scientific Psychology," "Personal Beauty and Racial Betterment," Etc. Price, \$3.50. Pp. 368. Illustrated. St. Louis: The C. V. Mosby Co., 1922.

Professor Dunlap has selected the topics for his chapters with some conservatism. Sense Perception, Thought and Thought Content, The Bodily Mechanism, Reaction and Consciousness, Instinct and Habit, Development of Perception, Space Perception, The Thinking Process, Affective Experience, The Empirical Self or "Me"—these are headings which we expect to find in any present-day text that claims to be orthodox. Concerning these conventional topics, however, Dunlap has written opinions which in many cases depart far from orthodoxy. Sometimes these opinions are quite original. They are always stated clearly and concisely.

The discussions in this book are concerned almost exclusively with conscious activities. It is not strange, therefore, that the first chapters are devoted to sense perception rather than to types of movement or some other supposedly more dynamic subject. We do not mean to imply that Dunlap's systematic position leans toward the static and structural. While his major interest is in processes that are conscious, he looks on this consciousness as something that is present under specified conditions of organic reaction. Mind, to him, is a social functioning of cells in the process of adjustment to stimuli. To this extent he is definitely in accord with the biologic type of functional psychology.

In considering the methods of psychology, Dunlap shows a strong liking for and faith in experiment. Certain advocates of introspection, who are also advocates of experiment, may be disturbed when they read this author's comparison of the two methods. Introspection, in practice, "very often seems to come down to the observation of all content, so far as possible, which is present during any given period of time. This method is sharply opposed to the experimental method, which always aims to narrow the observation at any given time to a small group or detail of content, selected in advance for observation, withdrawing attention for the moment from all other content." (p. 333.)

An important element in Dunlap's general position is his assumption that consciousness, while empirical fact, is not observable. The observable facts of psychology are the somethings of which we are conscious. Often such presuppositions, though interesting in themselves, do not affect a writer's more specific views. But that is not the case here. When he comes to consider imagery, Dunlap is not confronted with the usual problems that arise in this connection. An image is something thought of, rather than a process of thinking. Since we can think of colors or sounds or tastes, it is not difficult to account for different modes of imagery. The modality of an image is identified with something thought of, and, since the thinking of is not itself observable, naturally it can have no modality to be explained. At least, this is the way Dunlap's position impresses the reviewer.

Dunlap's analysis reduces the experienced world to *sentienda* (sense qualities?) related to each other in intricate ways. It contains nothing else. Feelings are particular organic or bodily *sentienda*. The relations existing between *sentienda* (or between other relations) are objects of consciousness. They can be perceived and thought about just as can *sentienda*. Some of the simpler relations are identity, difference and betweenness.

Dunlap thinks of habits as modifications of instinctive reactions. He criticizes McDougall and others for basing their classifications of human instincts on teleological considerations. In connection with affective experience, however, he proposes a theory of fundamental desires which does not differ markedly from McDougall's formulations.

An appendix to the book gives descriptions of certain abnormal conditions, that is, the psychoses, neuroses and amentias. This chapter is useful in itself, but it surely could be improved if the phenomena it describes were considered more explicitly in the light of habit formation and other fundamental concepts of functional psychology.

Taken as a whole, Dunlap's book is a worthy companion for the other serious texts in psychology. The style is straightforward and business-like. The student who works though it will not be amused, but, if he has caliber, he will be interested. There seems to be a general impression abroad that a textbook in psychology must be dogmatic. This one follows that rule to a considerable extent. Where the majority of facts or opinions supports a particular view, probably there is little danger in stating that view as *the view*. There are instances, however, in which Dunlap, like other authors subscribing to such a policy, states as fact opinions having scanty support. For instance, he describes the epicriticoprotopathic theory of cutaneous sensitivity as advocated by Head as established fact, whereas experimental evidence has rendered it extremely questionable. He has also stated the facts of color vision in terms of a theory—the three-color theory. Even granted that such a theory is the most satisfactory, how can the student of elementary scientific psychology be expected to keep a clear distinction between the theory and the facts? Much of the literature on color vision is needlessly difficult because of this practice of mixing fact and theory. As already indicated, however, such faults, to the extent to which they are faults, are shared with Dunlap by the run of writers of textbooks in psychology.

The publishers deserve credit for a fine piece of workmanship.

ANOMALE KINDER. VON DR. L. SCHOLZ. Dritte, Umgearbeitete Auflage von Prof. Dr. Adalbert Gregor, Direktor der Fürsorgeerziehungsanstalt Flehingen in Baden. Paper. Price, 72 marks. Pp. 312. Berlin: S. Karger, 1922.

As noted in the preface, this book commends itself especially to teachers of defectives and "special help" classes. It is written in popular style, explains the technical terms used and covers the subject matter in a satisfactory way. It should prove especially useful as a text for child-study clubs. The range of topics is indicated by the following chapter headings: Borderland of Mental Health; the Feebleminded; Nervousness; Hysteria; Epilepsy; Chorea; the Psychopaths; Disturbances during Puberty; Suicide; Treatment and Prophylaxis.

The problem of abnormal behavior in children raises so many issues, none definitely settled, that each page is a challenge to the critical-minded. Devia-

tions from normal behavior can be roughly ascertained only providing a standard of behavior for the different ages has been established. The author stresses this point when he speaks about the gradations between normal and abnormal and defines the healthy child as one corresponding in his behavior to the standard for its age. But such standards have not been established, and the "percentages of deviation" productive of abnormalities have not been ascertained. The author is thus forced to the traditional manner of handling the subject, regarding for example "the nervous child" as full-bloom an entity as Kraepelin's dementia praecox. As in most popular treatises on this subject, there is much speculation and much propaganda—for prohibition and eugenics. Unusual stress is laid on alcohol as a cause of feeble-mindedness and epilepsy. The author states, as though they were well established facts, that one third of all epileptic patients have a direct heredity of alcoholism, and that psychopathic children, through alcoholic indulgence, can be made epileptic. In the same manner is the rôle of masturbation exaggerated in the etiology of insanity. In the chapter on the feeble-minded, the contributions of American clinical psychology appear to be unknown, nor are any references made to the literature on delinquent behavior. Tests of intelligence are cited without any reference to standards. The author regards intelligence tests as having only mass-value and finds them of little or no use in clinical diagnosis. This judgment of the clinician is based on the findings of physical stigmas, on the appearance of the patient ("Does he look bright or dull?"), on his history, especially in regard to spontaneity of interest, laughter, sense of humor, attention, response to pain and to a series of "gross" intelligence tests. This typical attitude of the clinician is maintained more or less throughout the subjects enumerated. Its failings are obviously in respect to the fine gradations which the author points out in his introduction, and which require a more refined method of approach than clinical deduction. In the chapter on treatment, the rôle of prophylaxis is considered especially in regard to social reform movements. Behavior problems as such are not considered. Delinquent and truant behavior, unlike sex aberrations, are all treated as symptoms of mental aberration. This precludes what has come to be regarded as an important chapter in abnormal behavior—not necessarily the behavior of abnormal children, but behavior byproducts of our social and economic structure. There is again no cognizance of the vast American literature on the subject.

In spite of the shortcomings inherent in the popular presentation of this subject, the book remains one of the best of its type.

SYPHILIS OF THE INNOCENT: A Study of the Social Effects of Syphilis on the Family and the Community, with 152 Illustrative Cases, Made Under a Grant from the United States Interdepartmental Social Hygiene Board, by HARRY C. SOLOMON, B.S., M.D., Chief of Therapeutic Research, Boston Psychopathic Hospital, Instructor in Psychiatry and Neuropathology, Harvard Medical School, and MAIDA HERMAN SOLOMON, A.B., B.S., Research Social Worker, Boston Psychopathic Hospital, Boston. Washington: United States Interdepartmental Social Hygiene Board, 1922.

In attempting to review a book, it is important, first, to know its purpose. This is an intensive study of the family and social importance of syphilis. It was carried out under a grant made by the Interdepartmental Social Hygiene Board. An attempt has been made to present the subject of syphilis

in its social aspects portraying the practical problems as they actually arise, and also to show that they are of more than purely medical interest. The subject is treated, first, in relation to the individual; second, as concerning the mate; third, as affecting the child; fourth, as influencing the family, and fifth, in relation to the community.

The book is fully documented, and the study is based largely on observations made at the Boston Psychopathic Hospital and in that community. The old idea that an immune woman might give birth to a syphilitic child, under what has been called Colles' law, is conclusively shown to be without foundation; as is also Profeta's law that an apparently healthy child of a syphilitic mother could be nursed by its mother or a syphilitic wet nurse and yet not be infected. The employment of serologic tests has finally settled these questions.

The authors believe that there are different strains of the syphilitic organism, and they produce some case histories to support that contention.

Very properly, stress is placed on the necessity of making a careful family examination in any instance in which one member has been infected; the authors have found less difficulty in carrying out this investigation than is ordinarily apprehended by the medical practitioner. The authors properly attach more value to the clinical examination than to the result of the Wassermann test taken by itself. The question of the influence of syphilis on marriage is discussed at considerable length, without, however, reaching conclusions that are entirely definite. In the same way, legislation looking toward medical certificates of freedom from venereal disease is also set forth, with general approval of the intent of such law and full recognition of the difficulties of applying them, and with some suggestions for modifying them in the direction of uniformity.

Finally, the question of syphilis in the community is taken up from the financial standpoint, and an attempt is made to indicate in some degree the cost to the community of this disabling disease.

The book is prepared so as to be readily understood by the lay reader, but it gives such a general view of the disease in its medical and social relations that it is well worth reading by every medical man and every social worker.

THE PSYCHIC HEALTH OF JESUS. By WALTER E. BUNDY, Ph. D. Pp. 299. New York: Macmillan Co., 1922.

The author, associate professor of the English Bible in De Pauw University, sets forth to prove that the psychiatrists and neurologists who have written estimates of the mind of Jesus have held perverted views. An analysis of the "pathographers" whose views regarding the mental state of Jesus are cited, reveals no name of importance in the field of psychiatry. One is inclined, indeed, to doubt that any scientific psychiatrist would be greatly interested in the analysis of the writings attributed to Jesus for the purpose of forming an opinion as to his mental state. True, Freud and his disciples have read out of the lives of many great artists, dramatists and writers queer conclusions as to their sexual habits which do not seem to be borne out by any actual evidence; it might, indeed, be better to state that they have read into these writings more than the authors ever put there. But the problem of Jesus is a far more complicated matter. It may safely be stated that no man can approach the subject with the cold abstraction that scientific analysis demands; the authorities

whom Professor Bundy attacks so vigorously were not scientific psychiatrists but impassioned philosophers with some knowledge of psychology and diseases of the mind; and all of the men who oppose them, including Professor Bundy, were theologians, that is to say, believers whom one must convince against their will. Except as forms of mental exercise, such studies are as fruitless as the puzzles of Sam Lloyd. The modern psychiatrist will find a sufficient number of problems awaiting him in the etiology and treatment of mental diseases which continue to attack hundreds of thousand of living human beings in 1922. The essays on the psychology and mental health of the prophets of old may well be left to the theologians.